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TRAUMA INCIDENT TO SPORTS AND RECREATION

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BOSTON

THE wide interest of the American public in sports and recreation is apparent. The fact that the attendance at organized sports contests each year is over ten million indicates that this interest is real. The active participation in sports by over three million people portrays one aspect of the picture of modern America. Our daily press devotes pages of sports news in every edition to satisfy this craving of the public. Nor are these conditions confined to this continent. In England the association football, rugby and cricket teams play to enormous crowds and the number of participants runs well over three million.

Unfortunately, trauma occurs in sports and recreation in direct ratio of frequency and severity to the type of recreation chosen. Thus tennis and golf on the one hand, and skiing on the other, would scarcely be expected to be comparable in incidence of trauma; while among the organized sports, football and track would differ in incidence and severity of injury. However, what seems most significant in my experience with the college youth of today is that the most serious injuries encountered have not been in football but in unorganized skiing, in baseball and in polo.

Some three weeks ago our daily newspapers reported a grave injury to the president of one of our college outing clubs — a skiing accident involving a serious skull fracture. The same papers also reported that during an exhibition contest a popular professional ball player had fractured an ankle. Fifteen days ago an evening paper reported the removal from a White Mountain ski trail to a hospital of a member of a prominent ski club suffering with a broken leg. Whether such injuries were produced in sports competition or in recreation, the fact is obvious that they are altogether too numerous to wave aside as of little or no importance. Ever since the mountain ski trails were opened to the public, each winter has found more than one Harvard College student, recreation-bound, hospitalized in a New Hampshire hospital for weeks or even months, suffering from a fracture of one or both legs, a dislocated hip or some other serious injury. Nichols and Richardson,[1] writing in 1909, contended that medical supervision of a sport like football was a necessity and not a luxury. We all deplore this incidence of serious injury, whether it results from organized sports or from individual recreation.

Concomitantly with the injuries serious enough to be called to our attention by the press, there occur in even larger numbers those less serious injuries known under the diagnoses of sprains, strains and contusions. Only with a clear understanding of their pathology can come the complete comprehension of adequate treatment — aimed at the shortening of the disability period. In my experience the treatment generally prescribed by our colleagues for these less serious injuries seems to be lacking in knowledge of certain important pathological facts. Numerous as they are, perhaps a review in this modern era of trauma would be of interest.

Miltner, Hu and Fang[2, 3] have made extensive pathological studies of sprains experimentally produced in rabbits. Leriche (reported by Van der Linden[4]), Leriche,[5] Mirallié[6] and Arnulf and Freih[7] have recently drawn attention to the importance of the nervous system of joints. From this experimental work, together with my clinical experience with 638 cases of sprains, a definite picture of the pathology and of the tissue repair is here presented. First, there is the tearing of ligament and areolar tissue, which may or may not be associated with tearing of the joint capsule, and, accompanying it, hemorrhage. This hemorrhage infiltrates all adjacent tissue — tendon, areolar, extracapsular and intracapsular tissue and nerve fibers. Often the severity of the hemorrhage far exceeds that of the ligament tear and represents the damage done by tearing the capillaries and blood vessels in the periarticular areolar tissue. Secondly, from the hemorrhage and the col-

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lection of blood in and about the joint capsule there may develop an irritative intra-articular synovitis. Thirdly, from this bleeding develops a hematoma. Fourthly, by nature's process of healing the hematoma is absorbed; and fifthly, with the absorption, fibroblastic repair takes place. Thus we may summarize by saying that the tearing of ligament or periarticular tissues produces hemorrhage, which produces a local hematoma, that this hematoma must be absorbed by the natural processes of repair, and that eventual healing is by fibroblastic proliferation or fibrous scar tissue. Ligament tissue is particularly slow to regenerate, and through the fibroblastic repair a ligament loses its former elasticity and tone — "Once a sprain, always a sprain."

Table 1. *Number and Types of Injuries Received in Organized Sports at Harvard College for Five Years (Fall, 1932 - Spring, 1937).*

ACADEMIC YEAR	SPRAINS	STRAINS	SIMPLE CONTUSIONS	MUSCLE CONTUSIONS
1932-1933	173	57	84	121
1933-1934	179	94	122	102
1934-1935	97	87	34	126
1935-1936	100	85	29	101
1936-1937	89	60	44	115
Totals	638	383	313	565

(Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

Table 2. *Contusions Received in Organized Sports at Harvard College for Five Years (Fall, 1932 - Spring, 1937).*

	NO. OF CASES	
MUSCLE		
Thigh	282	
Legs	82	
Knee	46	
Shoulder	38	
Hip	35	
Back	23	
Arm	19	
Chest	17	
Buttocks	15	
Foot	5	
Head	4	
Hand	1	
Neck	1	
		568
JOINT		
Knee	142	
Ankle	34	
Shoulder	29	
Elbow	24	
Wrist	21	
Hand	20	
Hip	8	
Foot	4	
		282
SUPERFICIAL		
Leg	63	
Foot	56	
Hand	42	
Face	37	
Chest	27	
Back	19	
Head	13	
Neck	10	
Arm	10	
Hip	10	
Shoulder	8	
Thigh	5	
Testicles	4	
Buttocks	4	
Abdomen	3	
		311
Total		1161

In a recent monograph[8] I presented some interesting figures concerning the incidence of trauma in organized athletics at Harvard College. It may be of interest to review here such experience with contusions, sprains and strains. A total of 878 contusions, 638 sprains and 383 muscle or tendon strains have been observed among 6618 competing athletes (Table 1). All these injuries were of sufficient severity to prevent competition in sports for one or more sessions of practice or games and do not include trivial trauma. Similar in their pathologic pictures are all three conditions. All represent a tearing of tissue with concomitant hemorrhage, hematoma formation, hematoma absorption and repair by scar tissue. Whether produced by a sudden wrench or twist, a blow or bruise, or a pull or sudden muscular contraction, the specialized tissue is torn.

CONTUSIONS

In analyzing contusions it has seemed advisable to classify them as muscle, joint and superficial contusions. To Table 2 have been added approximately 280 superficial contusions which by their nature and location were not severe enough to interfere with sport sessions. It is of general interest to note that the thigh, leg and knee muscles are the most commonly involved in muscle contusions; that the knee, ankle, shoulder, elbow and wrist are the most commonly concerned in joint contusions; and that the leg, foot, hand and face are the commonest sites of superficial contusions.

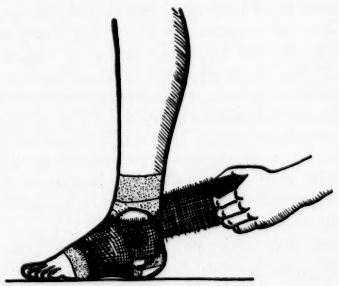

Figure 1. *The use of sponge rubber in a compression bandage for an ankle-joint contusion or sprain.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

The aim of treatment in all types of contusion

is to control or prevent hemorrhage, minimizing so far as possible the size of the hematoma. The smaller the hematoma, the shorter the period of its absorption, and hence the sooner complete recovery will ensue. Many contusions of joint, muscle and superficial tissue are sufficiently close to the integument to be effectively reached by the application of cold or ice water. Cold effects a closing down of the capillaries, and to be efficacious this treatment should be instituted at once. The injured part should be elevated during this phase of the treatment, which should be continued for at least an hour. The use of wet towels with

Figure 2. *The use of sponge rubber in a compression bandage for a knee-joint contusion or sprain.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

cracked ice between them is an effective method of application. After the hour of cold application, it is recommended that the contused joint or muscle be bandaged with a compression bandage; first wrapping with cotton wadding, next inserting a piece of 1-in. thick sponge rubber cut to fit the area, and finally winding firmly with an Ace or Bender bandage. Thus, in addition to the application of cold, one uses a bandage with compression to aid further in the control of hemorrhage. In severe contusions, rest of the injured part for twenty-four to forty-eight hours is essential in order to prevent a delayed hemorrhage. The entire early treatment is directed at the control of hemorrhage. Particularly is it of advantage to use sponge-rubber compression about joints (Figs. 1 and 2). The sponge rubber fitted to the part compresses not only the extra-articular tissues but also the joint capsule itself, and aids directly in limiting extravasation of intra-articular synovial fluid.

The later treatment, commencing after twenty-four to forty-eight hours, is intended to bring about the absorption of the products of hemorrhage or

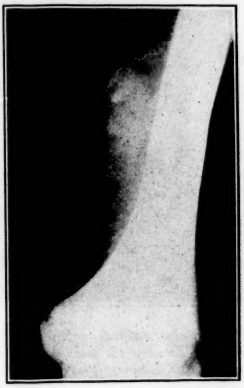

Figure 3. *Myositis ossificans traumatica in the triceps muscle belly (humerus).* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

hematoma. Agents which stimulate the lymphatics are important; they are applied proximal to the area of contusion, and day by day the area of contusion itself is gradually approached and finally included. It is a grave error to use these measures too early on deep-muscle contusions or joint contusions. This applies particularly to the application of heat to a contused joint and to vigorous massage of a deep-muscle contusion. These two agents, heat and massage, are the most desirable stimulants available for lymphatic absorption. Good effleurage and stroking massage are likewise aids to the maintenance of muscle tone.

Before leaving the subject of contusions, it is advisable to mention one definite complication of deep-muscle contusions, namely myositis ossificans traumatica. Such a complication occurs in severe deep-muscle contusions of the extremities, and it is diagnosed or suspected only when such a lesion fails to respond to therapy as well as might

be expected. It then becomes necessary to remove all massage or straining or friction from this area and to rely on heat alone for therapy. More than one case where this condition has been wrongly diagnosed as osteogenic sarcoma has been recorded in the literature. Kolodny[9] cites a striking example. Between fifteen and twenty-one days must elapse before x-ray can prove the diagnosis. Figures 3, 4 and 5 are illustrations of cases I have

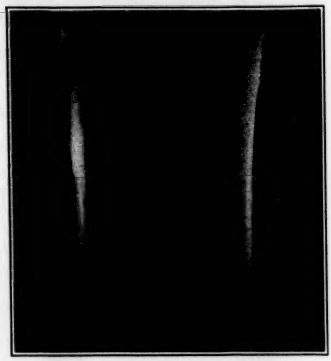

Figure 4. *Myositis ossificans traumatica resulting in a true synosteosis between the tibia and fibula.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

personally encountered. Simple myositis ossificans appearing along the shaft of a long bone always heals in the course of many months so that the x-ray can scarcely detect it, but if it occurs at the end of a long bone in the muscle origin or insertion near a joint, it will always interfere with normal function. In other words, we should expect a perfect functional end result when it occurs in the belly of a muscle, and permanently impaired function if it occurs near a joint (Fig. 6).

SPRAINS

Sprains are too often diagnosed without sufficient care of or attention to the individual ligaments involved. Whether a sprain involves one or more ligaments is a question of vital importance for a correct prognosis. Whether a sprain consists of a complete ligament tear or only a stretching is likewise significant. Which ligaments are involved, how many are injured and the degree of damage to each are essential facts to be ascertained at the earliest possible moment. However, I have made it a general rule not to give a prognosis until twenty-four hours after an injury, when a much clearer picture is unfolded.

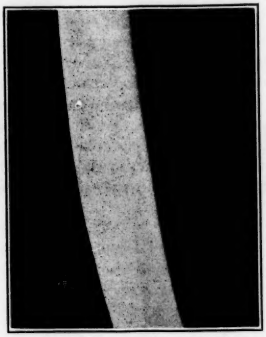

Figure 5. *Myositis ossificans traumatica occurring in the vastus intermedius muscle of the thigh.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

The diagnosis of a sprained ligament is made by careful digital examination over the course of all superficially palpable ligaments about a joint. Tenderness and swelling are the most valuable

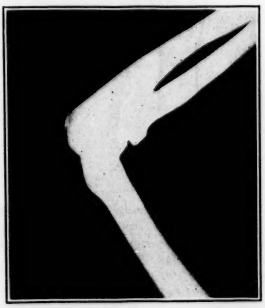

Figure 6. *Myositis ossificans traumatica about the elbow joint resulting in permanent impairment of function.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

signs. In addition, abnormal mobility of a joint in the lateral or anteroposterior planes is a most significant sign. Placing a gentle strain on a sprained ligament invariably elicits pain which the patient can sharply localize by digital

means. Perhaps a review by diagrammatic sketches will emphasize the points necessary to palpate in a sprain of the ankle or knee (Figs. 7 and 8). Time does not permit the giving of more details or the anatomical location of ligaments elsewhere in the joints of the body. If accurate diagnosis and accurate prognosis are the aim of modern surgery, this applies likewise in the field of lesser trauma.

Of perhaps even greater interest is the individual tabulation of the various ligament sprains of the ankle and knee joints (Tables 3 and 4). Here are recorded the incidence of involvement of the various ligaments in 332 sprains of the ankle and 200 sprains of the knee. It is startling to observe that in ankle sprains the anterior tibiofibular ligament is involved more than three times as frequently as any others, and that in knee sprains the medial collateral ligament is involved almost five times as frequently as any other.

The pathology of sprains being similar to that of contusions, in that one is dealing with torn tissues and concomitant hemorrhage from capillaries, the immediate treatment is similarly aimed at the control of hemorrhage. The application of cold or ice water with the part elevated, the use of cotton wadding, sponge-rubber strips and a compression bandage and rest are essential. The

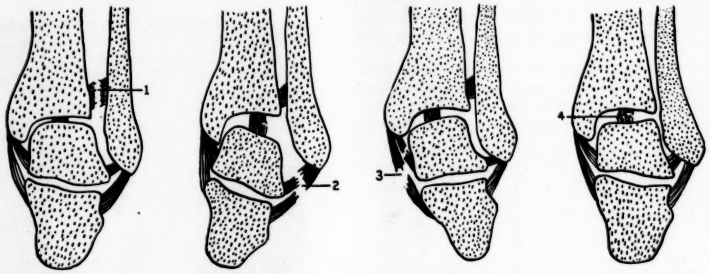

Figure 7. *Diagrammatic sketches of the four ligaments of the ankle that are commonly sprained: (1) anterior tibiofibular ligament; (2) lateral collateral ligament; (3) medial collateral ligament; (4) anterior tibioastragalar ligament.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

Table 3. *Incidence of Sprained Ligaments of the Ankle Received in Organized Sports at Harvard College for Five Years (Fall, 1932-Spring, 1937).*

Ligaments Involved	Academic Year 1932-1933	1933-1934	1934-1935	1935-1936	1936-1937	Totals
Anterior tibiofibular ligament	36	35	34	14	26	145
Lateral collateral ligament	11	0	8	9	13	41
Medial collateral ligament	3	4	6	5	3	21
Tibioastragalar ligament	2	6	0	5	2	15
Tibiofibular and lateral collateral ligaments	2	3	3	5	6	19
Tibiofibular and medial collateral ligaments	0	1	0	1	0	2
Tibiofibular and tibioastragalar ligaments	2	1	1	4	1	9
Lateral and medial collateral ligaments	0	0	0	2	0	2
Lateral collateral, medial collateral and tibiofibular ligaments	0	1	1	2	0	4
Lateral collateral and tibioastragalar ligaments	0	0	0	0	0	0
No ligaments mentioned	39	23	11	1	0	74
Totals	95	74	64	48	51	332

(Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

Figure 8. *Diagrammatic sketch of a sprain of the medial collateral ligament of the knee.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

principles which follow in the later treatment are the same as those already given concerning massage and heat.

It may be well to describe in a little more detail the treatment recommended for a badly sprained medial collateral ligament of the knee. Cold is applied at once and is continued for at least an hour. Cotton wadding, sponge-rubber strips and

Table 4. *Incidence of Sprained Ligaments of the Knee Received in Organized Sports at Harvard College for Five Years (Fall, 1932-Spring, 1937).*

Ligaments Involved	Academic Year 1932-1933	1933-1934	1934-1935	1935-1936	1936-1937	Totals
Medial collateral ligament	37	21	26	16	18	118
Lateral collateral ligament	4	2	7	8	4	25
Crucial ligament	4	4	2	0	0	10
Medial collateral ligament and medial meniscus	5	8	2	7	0	22
Medial collateral ligament and lateral meniscus	0	0	0	1	0	1
Medial and lateral collateral ligaments	0	2	0	1	2	5
Medial collateral and crucial ligaments	0	1	0	3	0	4
Lateral collateral and crucial ligaments	0	0	0	0	0	0
Lateral collateral ligament and medial meniscus	0	0	0	0	0	0
Lateral collateral ligament and lateral meniscus	0	0	1	0	0	1
Tibiofibular ligament superior	0	3	0	0	0	3
Patellar ligament	0	1	0	0	0	1
No ligaments mentioned	4	3	2	1	0	10
Totals	54	45	40	37	24	200

(Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

a compression bandage are applied about the knee, and the leg is encased in a light cast from calf to mid-thigh with the knee flexed at approximately 40 degrees. The patient is put to bed with his leg elevated on three pillows and remains there

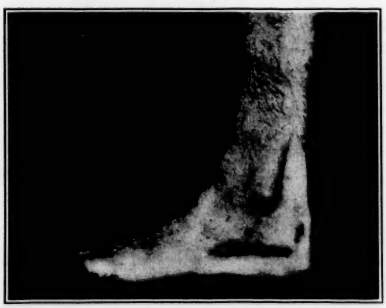

Figure 9. *The effectiveness of a rubber compression pad in controlling swelling in an acute sprain of the ankle.*

for forty-eight hours. The cast is then cut along its anterior surface and removed, and the inside bandage, sponge rubber and cotton wadding are removed. The knee will be found to contain no surplus fluid in the joint; the patient can flex it and extend it without pain; there will be swelling with actual ecchymosis in the skin, and tenderness over the torn ligament only. This use of compression and rest I find useful in severe ankle sprains as well. Through control of the hemor-

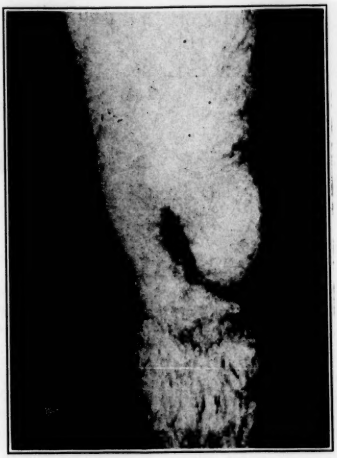

Figure 10. *Ecchymosis at the edge of a rubber compression pad, proving the value of the pad in the prevention of deep hemorrhage.*

rhage and compression of the joint capsule and torn tissues with sponge rubber and by rest, one minimizes the hematoma and swelling. The more rapidly hemorrhage can be brought to the surface, as shown in Figures 9 and 10, the easier it is to absorb or remove it with heat and massage. The shorter the disability period, the more grateful is the patient.

Too many physicians are strapping acutely sprained ankles and knees for the relief of pain. This is a grave mistake. Adhesive plaster on the skin surface under which deep hemorrhage is accumulating must be removed in a short while. The treatment of acute sprains is aimed at the control of hemorrhage. This is impossible to attain with adhesive plaster. The later treatment is directed toward a rapid return to function, and here the use of adhesive strapping is applicable only for later protection from recurrent sprains. In this connection it serves a most useful purpose. Once a ligament is torn or stretched it never regains its former tone. The joint fails to stand the strains of vigorous sport and must be supported by adhesive strapping. In our varsity, jun-

ior varsity and freshman squads, every ankle or knee sprained at any time from high school through the senior year of college is strapped

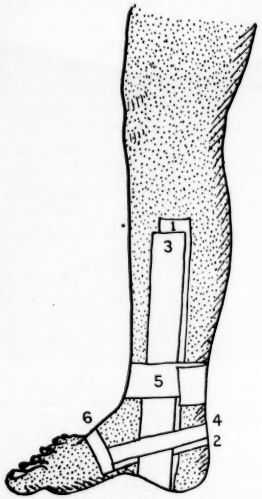

Figure 11. *Stirrup strapping for the prevention of recurrent ankle sprain.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

each day for all contact work. Recurrent sprains cannot be otherwise prevented. The prevention of recurrent injury is even more important than the proper treatment of acute injury (Figs. 11 and 12).

STRAINS

Of less interest to many, perhaps, are those injuries referred to as strains or pulls of muscle or tendon. Produced in many cases solely by an imbalance between protagonist and antagonist muscle groups, without outside wrenching or force, the lesion produced is nevertheless very real. The pathology is similar to that already described under sprains and contusions. There is a tearing of muscle or tendon tissue, and a rupture of capillaries and blood vessels, so that the concomitant internal hemorrhage may be massive, depending upon the degree of tear. The pathology of repair likewise is first hematoma formation, secondly hematoma absorption and thirdly healing by scar tissue. The hamstring muscles of the thigh show as clear an example of a true muscle pull as can be found. The sprinter's pulled tendon involves this area. The symptoms are striking and the end results of repair are reached tediously. The usual story is that a short, stocky sprinter, about half to three quarters along the distance of a 100-yd. or 220-yd. sprint, suddenly pulls up lame with an excruciating pain in the back of the thigh. His loss of function in the hamstring muscles is complete, and he has localized tenderness over the site of the tear, sometimes actually a palpable tumor. The treatment is the application of cold, followed by compression bandage and rest, until all danger of secondary hemorrhage has passed. I have encountered several cases where the hemorrhage was so massive that large areas of ecchymosis appeared later in the popliteal space, and still later around the Achilles tendon, descending by gravity and taking weeks to absorb.

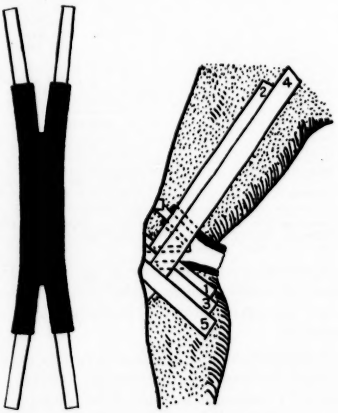

Figure 12. *The Duke-Simpson strapping for the prevention of recurrent knee sprain.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

The later treatment is aimed at the absorption of the hematoma by heat and massage in order to bring about the eventual restoration of function so far as is possible. In men of college age a partial tear of muscle or tendon is common. The repair is by scar tissue, and such muscle or tendon thereafter will never regain perfect function. It will, furthermore, be subject to recurrent strains or pulls at the site of the scar. In middle-aged adults such strains are more likely to be complete tears and often require surgery. One might wonder where these strains are most prone to occur (Table 5). It will be seen that among 375 mus-

cle strains almost 60 per cent occurred in the thigh and groin.

OTHER INJURIES

Among the more serious injuries of sports and recreation are fractures, dislocations and internal injuries. Time does not permit a graphic analysis of our experience. Suffice it to say that in my experience with 239 fractures in organized sports football produced the least serious ones. Baseball and polo have each produced one compound fracture, and hockey a badly comminuted leg fracture, and only these 3 cases required prolonged hospitalization. The incidence of serious fractures

Table 5. *Regional Incidence of Strains Received in Organized Sports at Harvard College for Five Years (Fall, 1932 - Spring, 1937).*

Region	Academic Year 1932-1933	1933-1934	1934-1935	1935-1936	1936-1937	Totals
Foot	1	7	1	4	4	17
Lower leg	9	9	11	5	2	36
Thigh	8	27	31	37	25	128
Groin	17	21	22	28	18	106
Abdomen	7	0	1	0	1	9
Buttocks	2	2	0	0	0	4
Hip	0	1	0	0	0	1
Chest	0	2	0	0	2	4
Back	10	7	13	7	5	42
Shoulder	2	11	5	4	0	22
Arms	1	1	2	0	1	5
Neck	0	0	0	0	1	1
Totals	57	88	86	85	59	375

(Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

in organized sport is therefore small. But with unorganized sports such as skiing, the incidence of serious fracture is all out of proportion to what it should be.

Dislocations, numbering 55 in my series, are even of lesser incidence. It is strange, however, that shoulder dislocations accounted for almost 50 per cent, and finger dislocations for 30 per cent. The most serious dislocations encountered were compound ones of the fingers, all occurring in baseball and all requiring the most scrupulous surgical technic. The treatment of uncomplicated dislocations should include an immediate x-ray, when available, followed by reduction, using the position of flexion in hinged joints. Shoulders are best reduced by the classic Kocher technic.

According to a five-year analysis of 3923 injuries produced in organized sports, internal injuries were relatively rare. Two injuries to the kidney, diagnosed by localizing signs and hematuria, were recorded. One hundred and six cases of mild brain concussion were encountered. In a recent study of 3 cases of brain concussion where routine lumbar punctures were made, not a single case showed increased intracranial pressure or microscopic blood in the spinal fluid. I believe that in any case exhibiting residual symptoms of amnesia, headache, dizziness or a lack of orientation as to time and place, the patient must not resume athletic competition or exercise until recovery is complete. The treatment, of course, is hospitalization, under careful observation as to pulse, blood pressure and general symptomatology. I believe in the appropriate application of dehydration with Epsom salts by mouth and rectum, 50 per cent glucose given intravenously after failure of the salts to relieve headache, and lumbar puncture in the more severe cases. Lloyd, Deaver and Eastwood[10] proved in their analysis of football deaths that the highest incidence of mortality occurred in sand-lot and high-school teams where medical attention to head

Figure 13. *Common site of hamstring pull.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

injuries was completely neglected. The ignorance of the laity of the serious complications that may follow a simple concussion of the brain is to be deplored.

The only other recorded case of visceral injury in our files occurred before this five-year study, and involved a ruptured spleen. In the fall of 1937 another contused spleen caused us some anxiety, but the patient recovered without surgery.

It is far better to suspect an internal injury wrongly and keep the patient under observation and medical care than to neglect a case in which the signs and symptoms are only slightly suspicious.

Figure 14. *Ecchymosis following severe hamstring pull.* (Reproduced from *Athletic Injuries: Prevention, diagnosis and treatment* [Philadelphia: 1938] by courtesy of the publisher, Lea & Febiger.)

Such are in brief the commoner types of trauma incident to sports and recreation. More and more individuals are participating in sports, and more contusions, sprains and strains are to be expected in daily surgical and medical practice. The prevention of recurrent sprains is the doctor's responsibility. The pathology and treatment of contusions, sprains and strains are similar. If there is one thought I can leave with you, I should like it to be an emphasis of the importance of early, appropriate and adequate treatment for the control of internal hemorrhage in these minor injuries. The smaller the hemorrhage, the smaller is the hematoma to be absorbed, the smaller the fibroblastic scarring and the shorter the disability period. A good end result depends on a prompt return to function with a minimum of scarring.

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QUANTITATIVE DIFFERENCES IN THE EFFECTS OF ALCOHOLIC BEVERAGES

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ALCOHOL is involved in a considerable proportion of fatal motor accidents. In Sweden the problem of limiting such fatalities has been met more directly than anywhere else. All the participants in every accident are subjected to a test to determine whether and in what amounts alcohol is present in the blood. Every traffic officer is provided with the means of getting a few drops of blood, and this sample is sent for analysis to the laboratory of Professor Widmark at the State University of Lund. The verdicts of the courts are largely based on these analyses. The adoption of similar tests in the United States is now widely advocated. The needle puncture of the ear or finger to obtain blood is simple, yet under our laws and customs it could scarcely be performed by anyone except a physician, and then only with the consent of the motorist. This difficulty could be obviated by determining the amount of alcohol in the deep expired air of the suspect. But every such method so far proposed places upon the police the responsibility for expert analysis.

An even greater difficulty is involved in the interpretation of the results obtained from analyses. The words "intoxicated" and "drunk" express extreme conditions which ordinarily require no blood analysis for their identification. With those to whom such terms apply there is no problem. It is the driver who is not obviously drunk and yet whose driving ability may be impaired by alcohol who constitutes the real problem. He may or may not be "under the influence of liquor." Even when the amount of alcohol in his blood has been accurately determined, the question still remains as to what extent, if any, in each particular case the driving ability is impaired. The amount of alcohol in the blood that may definitely render one man unable to drive safely may be without appreciable influence on another.

Here, then, is a medicolegal-scientific problem of challenging importance. How are science, law and common sense to be combined so as to diminish alcoholic motor fatalities? How can the motorist who is a moderate drinker be warned and rendered a safe driver? The problem may be resolved into two main questions, to which we shall offer answers. (1) Can there be established by law a figure for that concentration of alcohol in the blood which it shall be mandatory upon the courts to accept as involving the defendant in being "under the influence of liquor"? (2) Can there be established definite relations between the amounts of the various alcoholic beverages imbibed and the concentrations of alcohol in the blood that will be induced thereby?

The second of these questions has received comparatively little study; the emphasis has been put upon the concentration of alcohol in the blood for a diagnosis of intoxication. The motorist who wishes to avoid being adjudged "under the influence of liquor" has had little information to guide him in his drinking except his own feelings; and it is characteristic of alcohol to cause perversion of judgment. If there are definite relations between the amounts of alcohol consumed in various forms and under various conditions and the concentrations reached in the blood, this information should serve him as a guide. Moreover, if the evidence of witnesses establishes the quantity and type of alcoholic beverage imbibed, this knowledge should constitute precisely as valid proof that he was "under the influence of liquor" — or not — as does analysis of the blood.

SIGNIFICANCE OF THE CONCENTRATION OF ALCOHOL IN THE BLOOD

There is a tendency to impute to the blood tests for alcohol a diagnostic accuracy which they do not possess. This belief was summarized in a recent statement by Selesnick:[1] "Blood alcohol determinations can detect those degrees of intoxication which ordinarily escape the detection of competent physicians." But as a matter of fact, it is those concentrations at which diagnostic aid is most needed for which there is no precise correlation between the concentration of alcohol in the blood and the resulting symptoms, or lack of them.

The effects alleged to correspond to various concentrations of alcohol in the blood have been determined from clinical criteria. Large numbers of individuals have been adjudged intoxicated by

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physicians, police and other witnesses and the degrees of their intoxication have been correlated with the concentrations of alcohol in the blood.* Mere observational diagnosis of slight intoxication is admittedly uncertain; and chemical tests standardized on such a basis can be no more accurate than the criteria from which they were derived.

There is no question that for large amounts of alcohol in the blood the pharmacological effects, such as definite intoxication, coma and death, correspond closely to the concentration. But the same correlation does not hold for the finer gradations accompanying low concentrations. This is true also for the action of most drugs; the physician does not expect to obtain from every patient precisely the same effect from ¼ gr. of morphine. Individual differences are especially marked for drugs that influence temperament and judgment. These variations, well understood by the physician, are often misunderstood by the jurist and chemist, who expect precise quantitative physiologic responses. But even the physician is occasionally inclined to yield to chemical tests that relieve him of diagnostic responsibility.

At present the usefulness of blood tests for intoxication may be summarized as follows: (1) demonstration that the blood contains no alcohol is absolute proof of non-intoxication; (2) the finding of alcohol in the blood demonstrates that alcohol has been consumed; (3) more than 1.5 mg. of alcohol per cubic centimeter of blood indicates definite intoxication.

The chemical test is weak just where it is most needed, that is, for amounts of alcohol in the blood between 0.5 and 1.5 mg. per cubic centimeter. At or below 0.5 mg. there is no appreciable effect on anyone, and physician, layman and police can see that there is not. Above 1.5 mg. everyone is definitely affected and is so recognized by all. It is between these values that individual variations are most marked. One man of excitable temperament may show definite disturbances of behavior which should preclude him from driving an automobile when the amount of alcohol in his blood is as low as 0.7 mg. per cubic centimeter. Another of phlegmatic temperament may show no disturbance although the amount is above 1.0 mg. Investigators who have used the blood test extensively admit the diagnostic uncertainty of the methods for concentrations from 0.5 to 1.5 mg.[3] Moreover, it has been shown that for rats the concentration of sugar in the blood markedly influences the pharmacologic effect of alcohol;[4] if the same is true for man, and presumably it is, there may be not only variations in the response to alcohol as between different individuals, but also different responses at different times in the same individual.

In making a diagnosis of "under the influence of liquor" the physician is dealing with the individual patient and not with statistical generalizations. Any high value presumed to differentiate the sober and the definitely intoxicated would, if rigidly applied, lead to many acquittals where convictions should be made and a few convictions where acquittals should be made. Any attempt to use blood tests for precise diagnosis will lead inevitably to much legal quibbling with expert testimony, and will lead also to the eventual discarding of a useful diagnostic aid.

If the diagnosis of "under the influence of liquor" is to be made on the basis of a chemical test, it will be necessary to recognize these facts and to meet them. This can be done only by adopting as a criterion, not the value at which the majority are just on the verge of serious intoxication, but rather the value below which all men are reasonably sober. In other words, we must discard all consideration of physiological variations in the response to alcohol and establish an arbitrary legal criterion. From present knowledge we are justified in holding that anyone with a concentration of alcohol at or below 0.5 mg. per cubic centimeter is sober.† From present knowledge we are also justified in saying that an occasional individual with an amount of alcohol in the blood only slightly greater than 0.5 mg. may be so affected that he should not drive an automobile. The establishment of 0.5 mg. as the arbitrary dividing line between sobriety and an appreciable influence of liquor would enable the courts to put the burden where it belongs. It would rest, not on the physician, but on the motorist—and pedestrian. Hairline diagnoses and decisions, clinical, chemical and judicial, would be obviated. On such a basis many individuals tolerant to alcohol would be unnecessarily controlled, out of consideration for the susceptibility of a few. In view, however, of the extreme danger from the drunken driver, it seems only reasonable to provide a margin of safety to the public by demanding unquestionable sobriety from even the most susceptible.

An arbitrarily defined regulation for control of the motorist, such as is proposed here, is not new; the same legal principle is inherent in all speed regulations. To say that a motorist shall be subject to arrest if his blood contains more than 0.5 mg. of alcohol per cubic centimeter is entirely analogous to saying that he shall be subject to arrest if he drives faster than twenty-five miles an hour inside the city. In each instance an arbitrary limit is set for maximum practical safety.

*Harger, Lamb and Hulpieu[2] give a summary of extensive literature on this subject.

†This is the value now used in Norway.[5]

ALCOHOL INGESTED AND CONCENTRATION IN BLOOD

Numerous studies[6, 7, 8, 9] have been made to show the relation between the amount of alcohol ingested and the concentration attained in the blood, urine and alveolar air. From these studies it is now well recognized that the dilution in which the alcohol is drunk influences the concentration reached in the blood, and that the presence or absence of food in the stomach exercises an even greater influence. In the majority of these studies pure alcohol in various dilutions has been used; in some the alcohol has been given as whiskey and in a few as beer; rarely have comparative tests been made of the effect of the different beverages. This important point in the study of the correlation between the amount of alcohol ingested and the concentration reached in the blood has been largely neglected, but we have found that the form in which the alcohol is taken may influence the concentration in the blood to a greater extent than any other factor or condition.

As long ago as 1919 Mellanby[6] observed in animal experiments a marked difference in the rate at which the alcohol of whiskey and of stout were absorbed, even when diluted to the same concentration of alcohol. He observed that less intoxication followed from an amount of alcohol given as stout than as dilute whiskey. He concluded that "either whiskey contains something which hastens alcoholic absorption or, what is more probable, stout contains something which tends to delay absorption." He assumed that a colloidal substance in the stout was responsible for the slower absorption, but he was unable to duplicate the effect by adding gum arabic or starch to alcohol.

In Mellanby's experiments, dogs were used. The blood upon which the determinations of alcohol were made was taken from the skin capillaries. The amount of alcohol given was sufficient to produce 4.0 or more mg. of alcohol per cubic centimeter of blood, corresponding to extreme intoxication. Ingestion of this large amount of alcohol required considerable time for absorption, as shown by the fact that the concentration in the blood continued to rise for one hour in the case of whiskey and for two hours in the case of stout.

Mellanby found that in spite of the differences in degrees of intoxication exhibited by the animals to which whiskey and stout had been given, the maximum concentrations of alcohol finally reached in the blood were nearly the same. This finding is, we believe, due to the fact that the concentration of alcohol in blood from the skin capillaries falls below that in the arterial blood during absorption; the more rapid the absorption the greater the discrepancy.[10] It is unlikely that the amounts of alcohol in the arterial blood would have shown the same agreement. Instead they would have been much higher for the rapidly absorbed whiskey than for the slowly absorbed stout. The physiological effects from alcohol follow more closely the concentration of alcohol in the arterial blood than that in the capillary blood.[10]

We have repeated these experiments on human beings, using beer instead of stout, and gin as well as whiskey, and have determined the concentrations of alcohol in the blood from the alveolar air instead of from capillary blood. We have, moreover, used amounts of alcohol comparable, not with deep intoxication, but with moderate drinking. Under these conditions we find that the maximum concentration of alcohol in the blood induced by beer is less than half that from the same amount of alcohol as whiskey diluted to the same concentration. The difference between beer and gin is even more marked.

Numerous cases have come to our attention where motorists, obviously drunk, have stated in court that they had taken nothing but beer, often only one or two glasses. Such testimony, probably intended by the suspect to disprove his intoxication, has nevertheless been widely quoted as indicating that drunkenness can result from the use of beer. Our findings indicate that beer in reasonable amounts is essentially non-intoxicating. Carlson and his collaborators[9] have shown this to be true for beer containing 3.2 per cent of alcohol by weight.

Using the three common alcoholic beverages, gin, whiskey and beer, we have attempted to define as a rough guide the volumes of each which may be taken before and after meals that will not induce concentrations of alcohol in the blood above 0.5 mg. per cubic centimeter.

EXPERIMENTAL PROCEDURE

Numerous experiments were made on each of 5 young men ranging in weight from 150 to 175 lb. They were all moderate, occasional users of alcohol. The forms and times at which the various alcoholic beverages were taken are shown in Table 1. Each fifteen minutes thereafter the concentration of alcohol in the alveolar air was determined by analysis, and from this value the amount of alcohol in the arterial blood was calculated in milligrams per cubic centimeter. For the analyses the iodine-pentoxide method[11] was used. At the end of a forced expiration the exhaled air was passed by a side tube directly into the analyzer train; the volume of air was measured by displacement of water from a calibrated cylinder at the end of the train. No rubber or stopcock grease

was used in any of the connections of the train; for we have found that when the expired air is collected in a rubber bag as much as half the alcohol in the air may be absorbed by the rubber in the course of a few minutes. In converting the alveolar concentrations of alcohol into blood concentrations, we have used the distribution coefficient of 1:1150 established by Haggard and Greenberg.[11] The coefficient of 1:2000 used by Harger, Lamb and Hulpieu[2] can be shown by calculations from the chemical constants to be too high. We have compared the values obtained from alveolar air, using the distribution coefficient of 1:1150, with those obtained directly from blood drawn from the skin capillaries *after distribution of the alcohol in the body was complete,* that is, from two to three hours after ingestion of the amounts given. There was always precise agreement between the values so obtained.

Table 1. *Influence of Various Beverages in Different Amounts and Dilutions on the Concentration of Alcohol in the Blood.*

BEVERAGE: AMOUNT AND DILUTION	EQUIVALENT VOL. OF PURE ALCOHOL	COMMENTS	AVERAGE OF MAXIMUM BLOOD CONCENTRATIONS*	EXTREMES OF MAXIMUM BLOOD CONCENTRATIONS*	TIME AT WHICH BLOOD CONCENTRATION FELL TO 0.5 MG. PER CC.
	cc.		*mg. per cc.*	*mg. per cc.*	*min.*
GIN					
126 cc. 87-proof undiluted	55	Before breakfast	1.01	0.97-1.06	105
126 cc. 87-proof diluted to 1000 cc. with water	55	Before breakfast	0.92	0.87-0.95	86
63 cc. 87-proof undiluted	27.5	Before breakfast	0.54	0.57-0.51	—
126 cc. 87-proof undiluted	55	After breakfast	0.41	0.44-0.39	—
126 cc. 87-proof diluted to 1000 cc. with water	55	After breakfast	0.36	0.38-0.32	—
63 cc. 87-proof undiluted	27.5	After breakfast	0.27	0.31-0.24	—
WHISKEY					
122 cc. 90-proof undiluted	55	Before breakfast	0.89	0.91-0.85	135
122 cc. 90-proof diluted to 1000 cc. with water	55	Before breakfast	0.72	0.76-0.68	75
61 cc. 90-proof undiluted	27.5	Before breakfast	0.51	0.43-0.47	—
122 cc. 90-proof undiluted	55	After breakfast	0.35	0.37-0.33	—
122 cc. 90-proof diluted to 1000 cc. with water	55	After breakfast	0.32	0.35-0.28	—
61 cc. 90-proof undiluted	27.5	After breakfast	0.19	0.23-0.16	—
BEER					
1222 cc.	55	Before breakfast	0.44	0.46-0.39	—
1222 cc.	55	After breakfast	0.24	0.29-0.22	—

*Calculated from the analysis of samples of alveolar air.

In Table 1 are presented the results showing the maximum concentration of alcohol reached in the blood following the ingestion of the various beverages in the amounts and under the conditions given. This maximum occurred in most cases within thirty minutes after the beverage was taken. Thereafter the concentration fell gradually.

DISCUSSION OF RESULTS

It will be seen from Table 1 that on an empty stomach 63 cc. of 87-proof gin (containing 27.5 cc. pure alcohol) induces a maximum blood concentration of alcohol of 0.54 mg. per cubic centimeter, while 126 cc. of gin (55 cc. pure alcohol) induces a concentration of 1.01 mg. For 90-proof whiskey the values for 61 cc. (27.5 cc. pure alcohol) and 122 cc. (55 cc. pure alcohol) are 0.51 and 0.89 mg. respectively. With increase in the amount of spirits taken the alcohol in the blood does not rise proportionately. The relation is closer for gin than for whiskey.

It will be noted that dilution lessens to some extent the concentrations of alcohol reached in the blood. Although on an empty stomach 126 cc. of straight gin gives a maximum concentration in the blood of 1.01 mg., the same amount diluted to 1000 cc. with water gives a maximum of 0.92 mg. This decrease is less than 10 per cent. Under the same circumstances the same amounts of alcohol as whiskey give maximum values of 0.89 and 0.72 mg. respectively, a difference of nearly 20 per cent. The substitution of carbonated water for plain water to dilute the gin and whiskey had no influence on the concentration reached in the blood.

While gin is absorbed more rapidly than whiskey, that is it has a greater "kick," it is also eliminated more rapidly. After drinking 55 cc. of alcohol as gin, the alcohol in the blood rose to 1.01 mg. per cubic centimeter but fell to 0.5 mg. in 105 minutes; after drinking the same amount of alcohol as whiskey, the alcohol in the blood rose to only 0.89 mg. but did not fall to 0.5 mg. until 135 minutes later.

Food in the stomach (within one hour after a mixed meal, breakfast, of average size) greatly influenced the rate of absorption and consequently the maximum concentration of alcohol reached in the blood. The maximal concentrations induced by 126 cc. of 87-proof whiskey were 0.89 mg. before a meal and 0.35 mg. after one; for the same amount of alcohol as gin the corresponding values were 1.01 and 0.41 mg. A few experiments with

meals high in butter, olive oil and milk gave somewhat lower figures than for an average meal, but the results were not strikingly different.

Alcohol, when taken as beer, is absorbed very much slower than when taken as spirits, — or even as diluted spirits, — and the maximum concentration reached after a given amount of alcohol is lower. Thus while 55 cc. of alcohol as gin gave a maximum blood-alcohol concentration of 1.01 mg. per cubic centimeter and as whiskey 0.89 mg., the same amount as beer (1222 cc. of 4.5 per cent alcohol by volume) gave a concentration of only 0.44 mg. After a meal the maximum reached for the same amount of beer was 0.24 mg.

It has long been assumed that alcohol is readily absorbed from the stomach. Our findings cast some doubt on this belief. The rate of absorption, as judged by the maximum concentration reached in the blood, varies for the three beverages studied with the amount of buffer substances they contain. A trace of acid is sufficient to render gin acid; whiskey requires somewhat more; and for beer a considerable amount must be used. These differences suggest that the rate of absorption is influenced by the rate at which the beverage is passed into the intestines.

A few tests made with Martini cocktails showed that alcohol in this form is absorbed at about the same speed as gin.

The above values are for individuals weighing from 150 to 175 lb.; for weights considerably less or greater the figures for the maximum concentrations in the blood would presumably be correspondingly increased or decreased.

CONCLUSIONS

From the data presented it may be concluded that 60 cc. (2 oz.) of whiskey (a highball), somewhat less of gin (a Martini cocktail) or 1000 to 1500 cc. (1 to 1½ qt.) of beer may be taken on an empty stomach without the concentration of alcohol in the blood exceeding 0.5 mg. per cubic centimeter. After a meal, double these quantities can be taken with the same effect.

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A STUDY OF SEVENTY-FIVE TRANSFUSIONS WITH PLACENTAL BLOOD

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THE storage of blood with subsequent utilization is one of the newer advances in transfusion technic. The pioneer work with cadaver blood has been reported from the Russian hematological institutions in Moscow and Leningrad;[1] while Stavskaya[2] has recommended the conservation of placental and retroplacental blood, and Bruskin and Farberova[3] have used umbilical-placental blood. More recently Goodall and his associates[4] have proposed a technic for the collection of placental blood. With these reports in mind we have established a placental-blood bank at the Boston City Hospital, departing somewhat from the technic of the previous workers.

It is our belief that from a bacteriological standpoint the least intricate method of collection and storage is undoubtedly best. For receptacles we have selected 250-cc. and 500-cc. Erlenmeyer flasks, which are available in any laboratory. To these flasks has been added 15 cc. of 2.5 per cent sodium citrate, except for a few flasks where we used Citroseroid as the anticoagulant. A stopper consisting of a cotton plug covered with gauze is placed in each flask, which is wrapped together with a cork-stoppered test tube in a linen compress. These are autoclaved for thirty minutes and kept in a box to prevent tipping of the flask.

At the time of delivery two men must be scrubbed: one to act as accoucheur and the other to collect the placental blood after the baby has been delivered. The flask and test tube are unwrapped and handed without contamination to the assistant. The accoucheur holds the baby while the assistant doubly clamps and severs the cord 10 or 12 cm. from the umbilicus. A nurse palpates the abdomen to be certain there is not a multiple pregnancy. After the entire protruding cord has been painted with a sponge saturated with 3.5 per cent iodine, it is pinched between finger and thumb and 4 or 5 cm. of the distal portion is cut off with sterile scissors. A sterile sponge is lightly wrapped about the cord and held covering the mouth of the flask to prevent soiling from the vagina and rectum. With the cut end of the cord inside the flask, the digital pressure is released and the placental blood is allowed to drain. The cord is slowly milked as the blood drains, since we have found that a larger yield is obtained in this manner. Also during the process of collection the flask is rotated gently to admix the blood and the anticoagulating solution. After the blood has stopped flowing, the accoucheur finishes the delivery. Four to five cubic centimeters of the blood is transferred from the flask to the sterile test tube, and each is labeled in the delivery room with the mother's name and the date of collection. The flask is placed in an electric refrigerator kept at a temperature of 34 to 38°F. and is not disturbed until it is to be used. Flasks of identical blood groups are kept together in the ice box, so that we can determine how many bloods of each type are available without disturbing them.

The blood in the test tube is used in determining the red-blood-cell count and hemoglobin and for stained smears, and for making a cell suspension to establish the blood group.

Blood is taken from an arm vein of each mother at the time of delivery, and this is examined first by the "rapid" and later by the regular Hinton methods. Because of a recent experience, we have not relied on the tests on routine prenatal samples taken in the Out-Patient Department except to exclude the collection of syphilitic blood: a patient was referred from another Boston hospital for prenatal care; routine prenatal serological tests in both hospitals were negative, but a test on blood taken at the time of delivery was positive; further investigation disclosed syphilis in the husband contracted by him and, it is assumed, by the patient about the seventh month of her pregnancy. We have not used cord blood because of the frequent absence of a positive test in cases of congenital lues. Williams[5] states that only a small fraction of the children born of mothers with a positive Wassermann test present such a reaction at birth. Cruickshank[6] and Kilduffe[7] agree that the cord-blood Wassermann test is wholly unreliable. In 263 cases we have encountered positive Hinton tests in only 4; this low incidence of syphilis can be explained by the exclusion of patients whose bloods were found to be positive in the prenatal clinic.

One of the dangers of using placental blood for transfusion is the possibility of bacterial contamination. Bacteria may be present in the placental blood at the time of collection or they may be introduced during the procedure. In either case the con-

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taminating organisms are probably few in number and are usually killed by the living leukocytes in the freshly drawn blood. However, contamination from either source should be guarded against, and we now believe that a few drops of the citrated blood should be poured into a tube of plain bouillon (pH 7.6) at the same time as the sample is taken for the other tests. The flask should be immediately stoppered and remain so until use; failure of the bouillon tube to show growth following incubation for a suitable period of time is sufficient evidence for the sterility of the blood.

Table 1. *Average Blood Yield According to Sex and to the Weight of the Baby.*

Sex	Weight of Baby						
	under 5 lb.	5-6 lb.	6-7 lb.	7-8 lb.	8-9 lb.	over 9 lb.	all weights
	cc.	cc.	cc.	cc.	cc.	cc.	cc.
Males	52	79	104	105	115	127	107
Females	71	94	93	105	133	110	103

In this series, sterility was controlled by making aerobic and anerobic cultures on samples of blood that had been collected under identical circumstances as the other bloods and had been stored in the electric refrigerator for from one to thirty-six days. Twenty-six samples were tested, and in none were we able to demonstrate bacterial growth.

The average yield in 235 samples was 105 cc., with a variation of from 35 to 215 cc. The amount varied with the weight of the baby (Table 1), the length and size of the cord (longer and larger cords facilitated collection) and the speed of the technic (the faster the collection the greater was the amount). There was no essential difference between the yields from the cords of female and male babies of the same size.

Table 2. *Maternal and Fetal Blood Groups.*

Maternal Group	Fetal Group	No. of Cases
1	1	0
1	2	1
1	3	3
1	4	0
2	1	1
2	2	26
2	3	1
2	4	9
3	1	0
3	2	1
3	3	5
3	4	3
4	1	0
4	2	10
4	3	4
4	4	36

In a few cases we withheld pituitary extract, but the yield was no greater than where it was given immediately following delivery of the baby. Gentle massage of the fundus was advantageous as the placenta separated sooner with this procedure.

In 100 cases we found the fetal blood group varied from the maternal group in 33 (Table 2). In agreement with Goodall and his associates,[4] Polayes and his associates,[8] and contrary to Stavskaya,[2] we have demonstrated that babies have their own fixed blood agglutinogens at birth. We regrouped 50 babies before discharge from the hospital and found no change in group. The variance in the maternal and fetal groups was borne out in 5 multiple pregnancies. There were 4 sets of twins. Two were homologous, and in each instance the mother and both babies were in the same group — in one case Group 2 and in the other Group 4. Two sets were heterologous; in one instance mother and babies were all Group 2, and in the other the mother was Group 2, one baby (male) Group 4 and the other (female) Group 1. In a set of triplets the mother was Group 4, two of the triplets (identical males) Group 4 and the third (female) Group 2. As in adult blood, we have encountered subgroups in Groups 2 and 4, as evidenced by agglutination with several of the placental bloods in cross-matching with recipients, while there was no agglutination with other placental specimens of the same group. In every case where agglutination has occurred with bloods of the same group, we have retyped both placental and adult bloods in order to exclude an error in typing.

Hemolysis in some degree occurred almost routinely and was progressive upon continued storage. To substantiate this observation, red-blood-cell counts obtained at the time the stored blood was utilized were lower than counts at the time of collection. Counts as low as 3,000,000 were obtained, but the average was about 6,000,000 with a hemoglobin of slightly over 100 per cent (Sahli). The stained smears demonstrated anisocytosis and poikilocytosis and had a marked tendency to macrocytosis, with occasional nucleated red blood cells.

A definite advantage of placental blood is the absence of allergens, which according to Price[9] is a factor in curbing reactions. A questionable disadvantage might be the presence of analgesic or anesthetic drugs that pass through the placenta. Since analgesia is not used routinely at the Boston City Hospital, the possibility of the former did not warrant serious consideration. Ether was present in only a few samples: in 1 the odor of ether was unmistakable, and 2 recipients complained of its taste.

In this study, 75 transfusions were given to 46 patients, the largest number to any individual being 6. The indications (Table 3) were approximately the same as those for any transfusion, with, of course, a preponderance of gynecologic patients.

Before utilization the bloods were heated to body temperature. Next the gravity method of transfusion was used, with filtration of the blood through six to eight thicknesses of gauze in a funnel. As one blood ran out the next was added, with as little mixing as possible. Care was taken to prevent the mixing of the blood with saline or Ringer's solution, as clotting occasionally occurred when this was inadvertently done. Price[9] points out that this occurs when the solutions are hypertonic. The average amount of blood used was 325 cc., with the largest 525 cc., and the smallest 90 cc. The average number of bloods in each transfusion was three, with the smallest, one, and the largest, five. The average number of days the blood was preserved before use was six, with the longest, thirty days and the shortest, twelve hours.

Table 3. *Indications for Transfusions.*

	NO. OF TRANSFUSIONS		NO. OF CASES	
Infections		19		10
Septic miscarriage	5		3	
Septicemia	4		2	
Ulcerative colitis	4		2	
Pelvic inflammation	4		1	
Peritonitis (perforated ulcer)	1		1	
Ludwig's angina	1		1	
Hemorrhage		40		26
Leiomyoma	10		3	
Miscarriage	8		5	
Gastric carcinoma	7		4	
Gastric ulcer	3		3	
Perforated ulcer	3		3	
Postpartum hemorrhage	3		3	
Bladder ulcer	2		1	
Cervical carcinoma	1		1	
Ectopic gestation	1		1	
Premature separation of placenta	1		1	
Ruptured bladder	1		1	
Postoperative Treatment		14		8
Bowel resection	4		2	
Trachelorrhaphy	3		1	
Hysterectomy	3		2	
Ruptured uterus	2		1	
Radical mastectomy	1		1	
Tonsillectomy	1		1	
Miscellaneous		2		2
Aplastic anemia	1		1	
Pancreatic carcinoma	1		1	
Totals		75		46

Three patients experienced chills with a subsequent hyperpyrexia, which in two instances reached 101 and in the third 102°F. Another patient, who was afebrile, developed a temperature of 104°F.; this hyperpyrexia disappeared in twenty-four hours. Therefore, the incidence of reactions was 5 per cent. This is lower than the incidence of 11 per cent reported by Price[9] in utilizing fresh adult blood where an attempt had been made to eliminate the factor of diet and considerably different from that of 67 per cent given by Karavanov[1] utilizing preserved blood and that of 14 per cent obtained by Shamov[1] with cadaver blood.

Red-blood-cell counts and Sahli hemoglobin determinations were done routinely on all recipients during the forty-eight hours following transfusion. The maximum effect was obtained in thirty-six to forty-eight hours. There was an average increase of 400,000 red blood cells and 6.5 per cent hemoglobin following each transfusion.

Placental blood is an excellent substitute for blood lost by hemorrhage. It is also useful in preparing patients for operation. An excellent example of the latter was E. F., who entered the hospital with a moderate anemia due to a bleeding fibroid uterus. On admission the red-blood-cell count was 2,400,000 with 40 per cent hemoglobin (Sahli). She was transfused three times in nine days with a response which resulted in a red-cell count of 4,400,000 and a hemoglobin of 80 per cent. A similar case was M. D. who was in mild shock upon admission, due to hemorrhage from a concomitant fundal carcinoma and fibroid uterus. This patient was transfused preoperatively five times in nineteen days with a hematological response from 1,600,000 red blood cells and 26 per cent hemoglobin to 3,500,000 red blood cells and 55 per cent hemoglobin. She was bleeding actively on hospitalization and continued to do so until after she had been given two transfusions, which may have furnished a coagulating factor. Following a supravaginal hysterectomy, another transfusion was given, and the patient had an uneventful convalescence. Another benefit observed was an occasional pyretic crisis in septic cases. This was especially true in a patient having a bacteremia due to alpha-hemolytic-streptococcus septicemia. Following each transfusion there was a marked drop of temperature until the third transfusion after which there was no secondary rise; subsequent blood cultures were sterile.

Placental blood is always available for emergency transfusions. Four patients in the series were transfused on the operating table; in 2 of these cases we believe that the transfusion was a life-saving device.

SUMMARY

A technic for the collection and utilization of placental blood is described. The necessity for carefully typing and cross-matching placental blood is stressed as the fetal blood differed from maternal in 33 per cent of the cases. Seventy-five transfusions were performed on 46 patients, with the occurrence of minor reactions in 5 per cent of the cases.

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THE RELIEF OF CHRONIC BACKACHE AND SCIATICA BY MINOR SURGICAL MEASURES

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PHILADELPHIA

LUMBAGO and sciatica are everyday problems of the general practitioner. It is the present-day opinion that they form a combined condition, especially in chronic cases. The term "lumbosciatica" might well be used to describe this almost universal human ache. Pain in the lumbosacral region, as expressed in its nerve offshoots, is largely a penalty which man, a biped, pays for walking on his hind legs. The erector-spinae muscle group is in a state of contraction, whether one is sitting or standing, during all the waking hours. It is nature's law that senile fibrosis, with its accompanying pain, first appears in those structures hardest hit by life's wear and tear.[17] So the cartoonist depicts the old man not as holding his head with both hands but as supporting a lame back with them.

Acute lumbosciatica of the young man may be viewed clinically as a different entity, as regards cause and treatment, from the chronic condition of mid-life and old age. The acute condition of the young responds to medical treatment. The chronic condition of mature years requires minor surgical measures for its relief or cure.[1] These measures are now yielding such gratifying results that the pessimistic outlook of our medical forefathers toward helping this condition can be largely discounted, and the cartoonist may replace his doddering, bent old man with an erect and active one.

ACUTE BACKACHE

Acute backache does not differ from the pain attending increased intracellular tension in muscles and joints and around nerves in other body areas.[2] It is essentially an acute inflammatory swelling which may be localized in a definite muscle group or articulation or may center about a nerve trunk, but it is usually a combination of all three. In the lower back it commonly occurs as an acute lumbago or sciatica. Less frequently it occurs in the dorsal region as an intercostal neuralgia, which when accompanied by skin herpes is familiar to the laity as "shingles." More rarely it manifests itself in the cervical region as acute stiff neck involving the trapezius muscle or as neuralgia of the greater or lesser occipital nerves.

Acute backache as a rule results from muscle strain or exposure to wet or cold. Again, as in other body regions, it may result from foci of infection in the teeth, the tonsils or the gastrointestinal or genitourinary tract. The pain responds to rest, heat, elimination, analgesic drugs and counter-irritants. Its recurrence is guarded against by eliminating the toxic focus. This is essentially a self-curing condition, with Father Time acting as chief physician, while the medical man quiets the painful nerves and muscles by those soothing measures so familiar to us all. Minor surgical procedures are generally useless or else tend to aggravate the acute condition.

CHRONIC BACKACHE AND SCIATICA

The usual predisposing cause of chronic backache and sciatica is long-continued faulty posture. The upper spine tilts forward, with resultant traction on its related ligaments and attached muscles, strain of its articulations and irritation of its associated nerves. This posture is characteristic of age and of the obese younger individual with poor muscular development. In time the spine tends to partial fixation in its faulty position. Hartung[3] defines proper posture as one in which a plumb line dropped from the mastoid process bisects the tip of the shoulder, the hip joint and the knee joint, and strikes the foot just anterior to the ankle joint at the high point of the lateral arch. The head is up, the back is held straight.

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and the feet are not flat. Conversely, in poor posture the plumb line strikes the feet at or anterior to the heads of the metatarsals and the weight is thrown on the transverse arch. The head is held forward, the dorsal spine arches backward, the lumbar spine arches forward, and the abdomen protrudes; the knee joint is hyperextended, and the feet are apt to be flat. In popular phraseology the whole body slumps forward above the waist (Fig. 1).

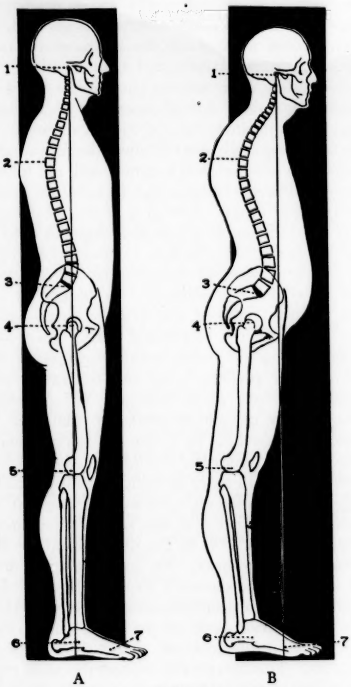

Figure 1. *A. Proper posture. A plumb line dropped from the mastoid process (1) passes through the tip of the shoulder, the lumbosacral articulation (3), the hip joint (4), the knee joint (5) and the base of the metatarsals (6). The head is up, shoulders back, dorsal and lumbar curves normal, knees straight and the lumbosacral articulation angle 43 degrees.*

B. Poor posture. The plumb line falls anterior to the lumbosacral junction and strikes the feet about the heads of the metatarsals. General body slump; that is, head forward, exaggerated dorsal and lumbar curves, protruding abdomen, exaggerated lumbosacral angle, hyperextended knees, feet with a tendency to be flat.

Flat foot[3, 4] and functional muscular insufficiency[5] are probably the most frequent causes of faulty posture. Rarer causes are conditions producing a permanent body list, such as a unilateral shortening of the leg from fracture or joint disease, or muscle-wasting from infantile paralysis. The usual immediate exciting cause is trauma, that is, muscle or ligament strain from an acute body twist, heavy lift or direct blow.

Contributory exciting causes are legion. They may be summarized as follows: referred pain arising from disease in other parts of the body, and direct pain arising from disease in or about the lumbar spine and its supporting structures.

Sources of referred pain are purulent foci from the teeth, tonsils, colon, gall bladder, prostate or uterine cervix; chemical or metabolic toxemias from lead, arsenic, alcohol, diabetes, gout or syphilis; diseases of the spine, as tuberculosis and metastatic cancer; diseases of the central nervous system, as tabes, meningitis and tumors of the lower cord or cauda; and pelvic abnormalities arising from the rectum or the genitalia.

Direct pain usually arises from anatomic variations or local degenerative changes about the fifth lumbar vertebra. Anatomic variations from the normal may be a transitional fifth lumbar vertebra,[13] a sacralization of the fifth lumbar vertebra,[3, 13] a defect in the laminae due to incomplete ossification of the fifth lumbar vertebra[13] or spina bifida.[3] Local degenerative changes usually result from an unstable fifth lumbar vertebra and may consist in any of the following: exaggerated lumbosacral angle;[13] spondylitis of the fifth lumbar vertebra;[13] narrowing of the intervertebral disk;[10, 11, 13] hernia of the nucleus pulposus into the spinal canal or into the body spongiosum;[9, 13, 14, 17, 18] narrowing of the intervertebral canal;[13] strain and subluxation of the posterior articulations;[13] sacroiliac strain;[6] fibrosis and contraction of surrounding muscles and ligaments;[6] fibrosis and contraction of the fascia lata;[8] fibrosis about and within the nerve trunks;[13] osteoarthritis of the posterior articulations and sacroiliac joints;[3, 15] pressure of arthritic deposits on the lumbosacral nerves;[3] or lipping of the vertebral bodies.[13]

Summarizing possible causes from the 2182 cases of authors quoted, one concludes (1) that any contributory cause may be important, but that few per se give symptoms without a superimposed acute trauma, or the chronic static trauma of poor posture; (2) that causes of chronic lumbosciatica whose frequency is exaggerated are toxic foci, constipation, hemorrhoids, prostatitis, female pelvic disease, especially uterine retroversion and retroflexion, anatomic variations in the fifth lumbar vertebra, sacroiliac strain or dislocation, osteoarthritis of the

young, the Marie-Strümpell type excepted, and true lumbar muscle fascitis; and (3) that the true cause is usually found in the spine itself, at or about the lumbosacral articulations, since the senile weakening of its ligamentous and muscular supports throws an extra strain on this vulnerable area and hastens the course of local senile fibrotic changes.

Faulty posture increases joint stress in proportion as it shifts the axis of weight-bearing anterior to a line passing through the lumbosacral junction. In normal posture the upper body weight is borne by the bony axis of the spine and the surrounding musculature tends to relax. A woman does not balance a load atop her head with the body bent forward but walks stiffly erect; that is, the spinal axis takes up the weight, and muscle and ligament tension is comparatively slight.

X-ray findings are apt to be negative. But if one finds a sacralization of the fifth lumbar vertebra, or narrowing of the intervertebral cartilages with lipping of the edges of the bones, or osteoarthritic deposits, one should bear in mind that the patient has probably sustained this condition without pain for many years; that "lipping of the lumbar spine is common after forty and universal after sixty";[8] and that, as has been said, "There is no such thing as a normal fifth lumbar vertebra."[3] Osteoarthritis in the young is rarely associated with back pain. In the aged it may aggravate the pain by blocking spinal flexion and extension. Or, in the event of an acute spinal bending beyond what the patient is accustomed to, a deposit may be chipped off and may cause extreme pain for some time.

Pathology

Lumbosciatica is not a fatal disease. I can find no autopsy report of microscopic findings in affected nerves. Our surmise as to local nerve pathology is largely drawn from results attending methods of treatment and the observation of nerve-root changes when exposed by laminectomy. That pain is due to a true neuritis is still a much debated question. It seems logical to suppose that in the long-standing case, continued low-grade irritation about the roots of the sciatic and gluteal nerves results in perineuritis, and finally endoneuritis with attending fibrosis, and that contraction of this fibrous tissue causes a diminished supply of blood to the affected nerve. "Pain is the cry of a suffering nerve for blood."[16]

That a true ischemic neuritis may exist is indicated by the following: relief from pain following methods tending to bring more blood into the affected nerve, for example, local heat, massage and active and passive exercises; relief from sciatic root pain following nerve stretching in the intervertebral foramina by epidural sacral injection or by postural, straight-leg flexion of the thigh; relief from sciatic trunk pain following endoneural injection of saline below the sciatic notch; the sensory and motor nerve reflex changes and the high protein content of the cerebrospinal fluid frequently found in advanced cases; Kimberley's[13] case of clinical perineuritis, in which, following laminectomy, he noted an absence of disk herniation, but an inflammatory injection of the nerves of the cauda, especially marked about the fifth lumbar root in the intervertebral foramen, with old inflammatory adhesions between the dura and the arachnoid; Fay's[20] 6 consecutive cases of clinical perineuritis, in which laminectomies were done for relief of chronic intractable unilateral lumbosciatica. Four of the latter showed disk herniation. In these there was direct pressure by the hernia on the fifth lumbar and first sacral nerve roots, where they rode over the protruding mass. There was erythremia of the nerve roots proximal to the hernia. The portions lying over the hernia and distal to it were much enlarged, and presented inflammatory congestion similar to that seen in acute conjunctivitis. In 1 case the affected nerve root was four times the size of its fellow on the opposite side. In a case in which air myelography suggested a herniation, laminectomy revealed an hour-glass constriction of the subarachnoid membrane about the roots of the cauda equina at the third lumbar interspace. No disk hernia was present. Another case showed a hypertrophied, partially calcified ligamentum flavum protruding 7.5 mm. into the posterior aspect of the spinal canal. The hypertrophied ligament had almost worn through the dura. The nerve roots showed the same type of congestion and enlargement as seen in the disk-herniation cases, but not so marked.

Symptoms

The pain or paresthesia of chronic lumbosciatica is transmitted through the lumbosacral and coccygeal plexuses, particularly by the great sciatic and gluteal nerve distributions. Classical symptoms in the mild case are initial muscle fatigue and morning back stiffness, followed by pain radiating over the sacroiliac joints and buttocks. Sciatica may precede but usually follows the backache. The pain is intermittent and is aggravated by unguarded movements, lifting, coughing, straining, straight leg-raising, exposure to cold and wet or a toxic focus. The majority of cases are in this class, and yield to palliative measures and minor surgical procedures. In severe cases of long stand-

ing, pain is constant. It is referred through the gluteal nerve over the buttock and sacroiliac joint and through the sciatic nerve down the back of the thigh and to the lateral aspect of the leg and the dorsum of the foot. Sensory and motor reflex changes develop. There is local tenderness on nerve pressure, contraction of the hamstring muscles and of the fascia lata, lateral list of the trunk, elimination of lumbar lordosis, fibrillary twitching of the buttocks, and muscular atrophy may occur. Finally there is almost complete limitation of lumbar spine motion by pain. This type of case usually requires a major surgical procedure for its relief.

Treatment

From every viewpoint chronic lumbosciatica is a complicated entity. "It is about as logical to refer all backaches to the orthopedic surgeon as it would be to refer all headaches to the neurologist."[3] The cause of lumbosciatica should be sought and in most cases treatment should be conducted by men of broad medical and surgical experience. The literature on this subject indicates that each specialty emphasizes the importance of its own field, and is exaggeratedly optimistic concerning results obtained by its own special methods. Operators[13, 19] of wide experience state that major operations are indicated in about 10 per cent of all cases and that palliative and minor surgical procedures are likely to give relief and restoration to functional usefulness in the remainder.

MAJOR SURGICAL METHODS

The usual operative procedures are fasciotomy, spinal fusion and laminectomy. A full discussion of them is beyond the limits of this paper. Fasciotomy of the fascia lata and its intramuscular bands has the advantage of being a relatively simple operation. No really satisfactory explanation has been offered for the results obtained. It gave relief in 73 per cent of 415 reported cases.[8] Spinal fusion or therapeutic bony ankylosis of the posterior lumbosacral articulations is the major operative procedure of choice in severe cases of long standing.[13] The immediate relief from pain is probably due to the division of the sensory nerves, as the origin of the gluteus maximus and the deep fascia are freed from the posterior aspect of the sacroiliac joint. This operation permanently relieves nerve-root pain, as does spinal fusion in tuberculosis or fracture of the spine; that is, complete elimination of motion puts at rest the overstrained muscles and ligaments and allays inflammation about the articulations and intervertebral foramina. It gave relief in 80 per cent of 247 reported cases.[19, 14, 13] Laminectomy is indicated where air myelography shows a disk hernia which persists on spinal extension, or in other conditions causing nerve-root pressure, for example, tumor, hypertrophied ligamentum flavum or inflammatory adhesions.[20] To eliminate the possibility of an unstable spine, laminectomy should be accompanied by spinal fusion.

PALLIATIVE AND MINOR SURGICAL METHODS

The patients in this relatively large group, in which palliative and minor surgical procedures are likely to give relief, too often accept their lameness with the resignation accorded wrinkles and gray hairs. Many seem satisfied with the most perfunctory treatment. They fail to realize that, unaided, the condition is surely progressive, but that with proper medical guidance they may have many years of comfort before them. A short period of confinement to bed is always most satisfactory. But many patients cannot for economic reasons enter a hospital, and may be treated successfully as ambulatory cases. Prolonged wearing of a cast, while at times necessary, usually means a long period of rehabilitation, owing to bone and muscle therapy.

As in every chronic condition, the first requisites of treatment are an accurate history and a thorough examination — physical, laboratory and x-ray. One should also remember that although the elimination of a toxic focus or malposition may not eliminate pain, it may prevent its recurrence following relief by successful local measures.

Elimination of Etiologic Factors

Toxic foci or referred pain. Despite the fact that chronic cases in the aged rarely yield to any but local treatment, we are all familiar with the occasional miracle which follows the removal of an abscessed tooth or infected tonsil, or the replacement and support by pessary of a retroverted, boggy uterus.[21, 22] So that every possible systemic or distant local focus should be put to rights.

Correction of faulty posture. This is best done by active exercise. The patient should be put through a course of spinal and hip flexions, extensions and torsions morning and night, to strengthen gradually the associated spinal muscles and to loosen ligaments and nerve roots. Those bending exercises which tend to give pain are the ones to be persisted in. The elderly individual must be persuaded to resume the setting-up exercises of his youth. This is perhaps the most difficult part of the whole regime to enforce, for it means a bit of self-sacrifice and a little real work in the naturally self-indulgent. But once the pa-

tient becomes interested in golf, for example, half the physician's problem is solved.

Correction of Flat Foot. This is one of the most important points in the correction of faulty posture. The flat-footed individual naturally slumps forward. With a little spring in his feet restored by shoes correcting lateral and transverse drop of the arches, the shoulders tend to go back. Why are high heels popular with women, aside from their making them supposedly more attractive? Because if the heels are elevated the shoulders must go back in order to preserve a proper balance (Fig. 2). Woman, with her weaker musculature, finds that she has less backache and fatigue at the end of the day when wearing a moderately high-heeled shoe with a broad walking surface.*

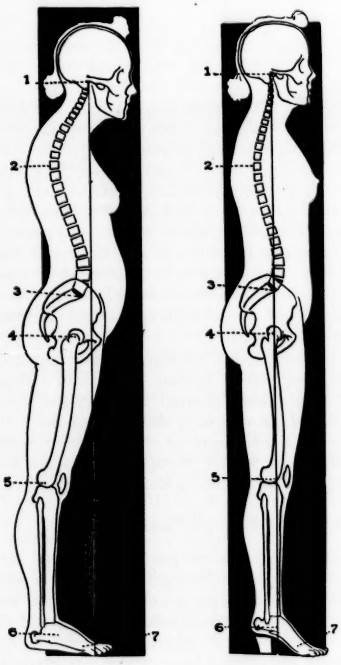

Figure 2. *A. Poor posture in a woman: weak musculature, heels flat.*

B. Improved posture by wearing moderately high-heeled shoes with broad walking surfaces. In the slumped figure, if the heels are elevated, the shoulders must go back to preserve a walking balance.

Measures to Absorb Local Fibrosis

Irrespective of the means employed, the object of the treatment of any local inflammatory condition is to bring more blood to and through the part. Practically the whole field of modern physiotherapy is based on this principle. More blood is brought to the affected area by heat and massage, passive motions and nerve-stretching. The administration of iodine in appropriate form still holds its place as a method for promoting the absorption of fibrous tissue.

Heat and massage. Throughout history heat in some form has been used locally to allay the pains associated with chronic inflammations of muscles, joints and nerves. Massage has been employed for the same purpose by all primitive as well as civilized peoples.[23] Heat of any kind—sunshine, electric pad, hot lamp, cold lamp or diathermy—holds its place as a means of temporary relief, and the hot flatiron applied over the family ironing blanket still has its adherents in rural communities.

Passive motions. Those of the hip and the pelvic and spinal joints have been largely responsible for giving our osteopathic confrères their success in the relief of back and sciatic pain. They should be used with caution in cases of advanced vertebral lipping, and are contraindicated where x-ray films show any degree of fracture dislocation, a lesion suggestive of carcinomatous or tuberculous disease of the spine, or disk herniation. Their value lies in relieving a partial false ankylosis of the spinal segments and in freeing adhesions of the spinal nerves in the intervertebral foramina.

Bankhart[24] indicates four manipulations under full anesthesia. We have found the intravenous injection of Epival satisfactory for a full course of manipulations, while the inhalation of ethyl chloride is sufficient for stretching of the sciatic nerve. Bankhart's technic is as follows:

1. Sciatic nerve-stretching. The patient lies on his back and an assistant steadies the pelvis. The operator holds the leg on his shoulder and keeps the knee stiff with one hand while lifting the leg to the limit of thigh flexion and swinging it back and forth several times with the other.
2. Spinal flexion. The patient lies on his back. The operator put an arm under both knees and brings them up to the head.
3. Spinal rotation. The patient lies on his back. An

*Fifty women between the ages of thirty-five and fifty-five were questioned by us as to the type of shoes worn during their daily work. All wore some type of lateral arch support. Sixteen wore a metatarsal pad, and 46 a heel more than 1 in. high. The average heel height was 1¾ in. The average heel width on the walking surface was 1⅞ in. Forty women (80 per cent) stated that wearing sneakers or shoes with low heels for any length of time caused discomfort or pain in the feet, calves, back of thighs and lower back. Most young women seemed indifferent to the type of heel and wore with comfort whatever the occasion demanded.

assistant holds down the right pelvis while the operator, standing on the left side, reaches over and forcibly pulls the right arm toward him. The procedure is repeated on the opposite side. Adhesions are often heard to crack during the manipulation.

4. Spinal hyperextension. The patient lies on his face. The operator puts his arm under the thighs above the knees and forcibly lifts the thighs with one hand holding the back down with the other.

With the exception of sciatic nerve-stretching, daily active motions carried out by the patient seem clinically to give more lasting results.

Nerve-stretching. This is accomplished by one of three different procedures.

Manipulation. The modern closed method of nerve-stretching has been described under passive motion. Forty years ago the open method of stretching, first carried out on the sciatic nerve, gave such gratifying results that it was used on all large nerve trunks of the upper and lower extremities. The trunks were picked up on the hook of a weighing scale, and tables were made out to show the proper number of pounds stretch to put on each trunk. Since we have learned that it is not the peripheral stretch but the drag on the sciatic spinal roots in the intervertebral foramina that does good, it is easy to understand why the open method has been largely abandoned.

Intraneural injection. This is the injection of fluid into the nerve sheath by skin puncture without exposing the nerve trunk by incision. The sciatic nerve, because of its large size and accurate localization, can be successfully injected. Intraneural injection results in a localized, fusiform swelling which stretches the surrounding nerve sheath and tends to separate the individual nerve bundles inside it. In experiments conducted by us it was found that injection of the sciatic nerve on a cadaver brought out the following points. In the sacrosciatic foramen the nerve rests on the spine of the ischium at an average depth from the skin of 5 cm. The skin-puncture guide is a point one fingerbreadth lateral to the medial third of a line drawn across the buttock from the sacrococcygeal junction to the inferior tip of the great trochanter. The nerve is lifted off the spine of the ischium for 0.5 cm. by the gemelli muscles. The nerve trunk is 2 cm. in lateral diameter and 1 cm. in anteroposterior diameter. Two cubic centimeters of fluid swiftly injected into the nerve sheath causes a fusiform swelling about twice the size of the normal nerve. Excess fluid quickly leaks out through the needle puncture holes into the surrounding tissues (Fig. 3).

The patient lies on his abdomen. The injection site is determined, and the skin is wiped with iodine and alcohol and anesthetized with ethyl-chloride spray. A 20-gauge spinal needle 8 cm. long is plunged in vertically, and strikes bone — the spine of the ischium — at an average depth of 5 cm. Pain usually shoots down the back of the leg as the needle pierces the sciatic nerve. The needle is withdrawn 0.7 cm. to ensure its point's lying inside the nerve sheath. Next, 2 cc. of 1 per cent novocain in saline solution is forcibly injected twice at one-minute intervals. Pain shooting down the back of the leg at the time of injection is clinical evidence that the needle point lies inside the nerve sheath. Failure will result if the injection is given extraneurally into a high nerve bifurcation. It is our practice to inject the nerve, wait a few minutes until the patient assures us that all sciatic pain has disappeared, and then do a postural nerve-stretching under ethyl-chloride inhalation anesthesia. The patient is sent home to bed for twenty-four hours with local heat applied to the site of injection. Results in 15

Figure 3. *Regional anatomy of sciatic nerve in the sacrosciatic notch. The surface landmark is a point one fingerbreadth to the lateral side of the medial third of a line drawn from the sacrococcygeal junction to the lower point of the great tuberosity of the femur. The deep landmark is the spine of the ischium.*

A. Size and position of the sciatic nerve as it lies on the spine of the ischium.

B. Fusiform swelling of the nerve: twice normal size following the intraneural injection of 2 cc. of fluid.

C. Needle plunged vertically into the soft tissues at the surface landmark.

cases during the past year were complete relief in 8, partial relief in 3, and failure in 4. Injection should never be employed in acute, fulminating sciatica.

Epidural sacral injection. It has long been recognized that an epidural injection of 30 to 60 cc. of a 1 per cent novocain solution into the sacral canal to produce sacral anesthesia is often followed by permanent relief of a chronic low backache or sciatica. Various authors advocated this method. But the research work of Evans,[25] of the London Hospital, in 1930 gave us our first true understanding of the rationale of the procedure. He showed that when 30 cc. of fluid was injected epidurally in the cadaver a true nerve-root stretching resulted. To appreciate his finding, let us look at the anatomy of the spinal sheath and its nerve offshoots in the lower lumbar and sacral spine.

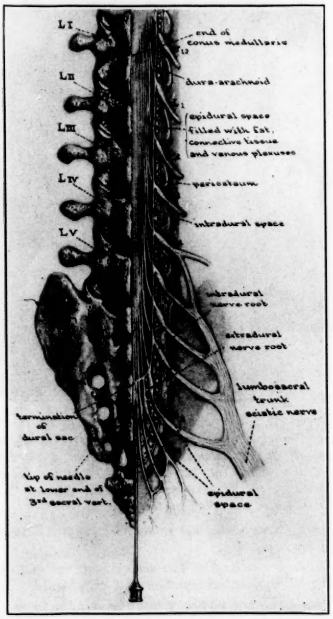

Figure 4. *Anatomy of epidural space: anteroposterior view. The space is filled with fat separating the dura from the intraspinal periosteum for the length of the spinal canal. The epidural space at the second, third and fourth sacral segments is of large size. The spinal nerves run in this space for a distance of 2.5 to 3.0 cm. from the point where they pierce the dura until they enter the intervertebral foramina. The needle piercing the dense sacrococcygeal ligament for a distance of 4 cm. is well below the dural sac.*

The spinal cord proper ends as the conus medullaris at the first lumbar vertebra. The dural sac continues down to the lower border of the second sacral vertebra. The sac contains cerebrospinal fluid and the cauda equina, that is, the lumbar and sacral nerve roots. Between the dural sac and the periosteum is the epidural space filled with semifluid fat. The dura is held ventrally by fine

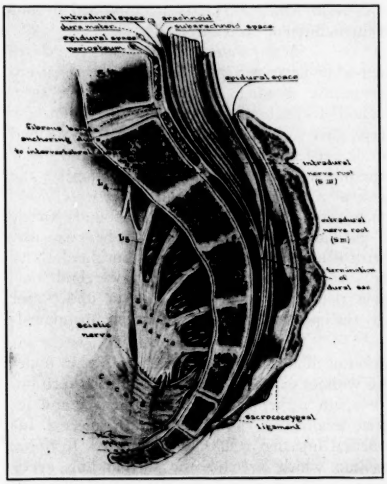

Figure 5. *Anatomy of epidural space: sagittal section. The anterior spinal roots enter the intervertebral foramina and emerge into the pelvis to form the sacral plexus. Fibrous bands anchor the dura anteriorly to the intervertebral disks. The dura is free posteriorly and laterally.*

fibrous bands which anchor it to the intervertebral disks. No bands bind it laterally or posteriorly. The space is prominent posteriorly at the second, third and fourth sacral segments. The space is prolonged upward in the spinal canal to the cervical region. The nerve roots run in this space for a distance of 2 to 4 cm. from the point where they pierce the dura until they enter the sacral foramina. The nerve roots carry a sleeve of dura out into the intervertebral foramina (Figs. 4 and 5).

Evans's work on cadavers and living subjects showed that injections of fluid into the epidural space (Fig. 6) had the following results: 30 cc. of fluid filled the sacral space; when 100 cc. of fluid was injected it could be traced into the cervical region; the fluid filled the space posteriorly

and laterally and worked out into the intervertebral foramina between the dural sleeve and the periosteum; the injection forced the dural sac upward 3 mm.; the slow, intermittent injection of 100 cc. of fluid gave a slowly repeated nerve-stretching and tended to separate adhesions of the nerve in the intervertebral foramina; and in the living subject a rise in spinal pressure of from 16 to 30 mm. was registered on a manometer,

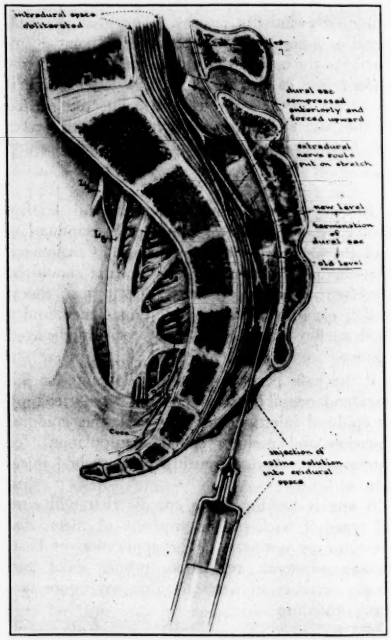
subdural space obliterated
dural sac compressed anteriorly and forced upward
subdural nerve roots put on stretch
new level
harmonium of dural sac
old level
Coccyx
injection of saline solution into epidural space

Figure 6. *Effect of injection of 30 cc. of fluid into the epidural space. The sacral nerves and whole spinal dural sheath are compressed anteriorly and the dural sac forced upward for 2.5 mm. This results in an intraspinal stretching of the nerve roots and tends to free adhesions in the intervertebral foramina.*

through a needle introduced into the fourth lumbar interspace, as the whole spinal sheath was forced upward and compressed. A true intraspinal stretching of the nerves follows intradural injection. The findings in our experiments on cadavers coincided with those of Evans.

For the injection the patient lies prone on the table with a narrow hard pillow under the suprapubic region. The sacral cornua are felt for. The hiatus, covered by the dense sacrococcygeal ligament, lies between the cornua and approximately 5 cm. from the tip of the coccyx. The skin is wiped with iodine and alcohol and anesthetized through a fine needle with 1 per cent novocain. This needle is pushed down through the sacrococcygeal ligament. A 20-gauge spinal needle 8 cm. long is pushed almost vertically through the ligament until it strikes bone, when it is slightly withdrawn; the butt is depressed and it is pushed up into the canal for 3 cm. The plunger of the empty syringe is pulled out, and if blood or cerebrospinal fluid appears, the needle is withdrawn until nothing is obtained on aspiration, 0.5 per cent novocain in saline solution is then slowly injected. The fluid should flow freely; if there is obstruction to the piston the needle point is probably imbedded in the periosteum. It is slightly withdrawn and the fluid is injected slowly and intermittently up to the point of referred pain, 100 cc. of solution being used. Pain along the course of the affected nerve is a favorable sign. Swelling over the dorsum of the sacrum is evidence that the needle is outside the sacral canal. The patient rests on the table for half an hour and is then sent home to bed, local heat being applied over the site of injection for twenty-four hours. Local heat, exercises and indicated general systemic treatment are mandatory after all minor surgical procedures. Table 1 gives results in 60 epidural injections given during the last three years.

Table 1. *Results of Epidural Injection in 60 Cases.*

LOCATION OF PAIN	CAUSE	NO. OF CASES	NO. CURED OR MARKEDLY RELIEVED	NO. OF FAILURES
Buttock and sacroiliac region	Idiopathic	15	15	0
Sciatic nerve	Idiopathic	16	10	6
Coccyx	Traumatic	12	12	0
Lower dorsal and upper lumbar region	Idiopathic	4	3	1
Lower back	Cancer of prostate	2	0	2
Lower back	Cancer of uterus	1	0	1
Lower back	Cancer of breast	2	1 (5 mo.)	1
Lower back	Tuberculosis of mid-dorsal spine	1	1	0
Amputation stump	Endarteritis obliterans	7	0	7
	Totals	60	42	18

In 170 reported cases of sciatica,[25, 26, 27] including 16 of our own, cure or marked relief resulted in over 70 per cent. It is worthy of note that in our 60 epidural injections complete relief of back pain occurred in all the cases of coccydynia type and in those of the sacroiliac type, with pain radiating down over the buttocks. It is our belief that epidural injection offers more hope of relief to patients with chronic low back pain and sciatica, who have been for months or years through the usual therapeutic mill, than any other form of medical or minor surgical treatment.

Paravertebral Alcoholic Injection

This should be used only in cases with disturbances involving purely sensory nerves or mixed nerves with an unimportant motor supply. It is the treatment of choice in chronic intercostal, great occipital and lesser occipital neuralgia. Perineural

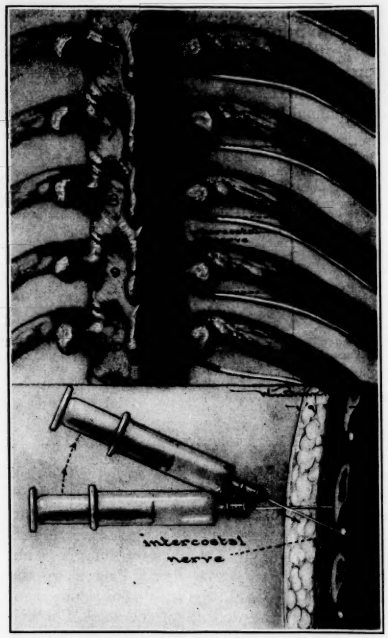

Figure 7. *Anatomy of intercostal nerves. They enter the costal groove at the palpable, posterior rib angle, 6 cm. lateral to the spinous processes.*

Plan of alcohol injection. The needle should first strike the lower edge of the rib. Then the point is depressed until it slips by the rib edge and strikes the nerve. The intercostal artery and nerve lie above the needle point, protected by the rib.

or endoneural injection with absolute alcohol gives complete sensory anesthesia for an average time of two months.

The painful intercostal space can be accurately located by the patient and checked by deep finger pressure 4 cm. lateral to the spinous process. The nerve enters the intercostal groove 6 cm. from the mid-spine. At this point the posterior angle of the rib is palpable. A 23-gauge hypodermic needle 3 cm. long, attached to a 2 cc. syringe, is inserted until it strikes the lower edge of the rib; the needle point is worked down until it slips by the lower rib border (Fig. 7). The patient complains of a shooting pain along the lower rib border when the nerve is touched. Two cubic centimeters of 2 per cent novocain is injected. In two minutes 5 to 8 cc. of absolute alcohol is injected. Even with the primary novocain, the pain may be intense for one to three minutes, but is followed by complete analgesia along the nerve trunk. The anesthesia persists for from one to three months. Normal sensation eventually returns. The great occipital nerve is accessible for injection at a point 3 cm. lateral to the occipital protuberance, at the superior curved line of the occiput. The lesser occipital nerve lies on the same transverse line just medial to the base of the mastoid process. Both nerves can be accurately located by firm finger pressure.

Summary

Patients with chronic backache and sciatica should be subjected to a thorough examination, and any toxic focus, systemic disease or malformation should be treated. Faulty posture should be corrected by active exercises and proper shoes; golf is recommended for middle-aged men, and a moderately high-heeled shoe for middle-aged women.

If the pain persists, use the following: for sciatica, endoneural injection, postural nerve-stretching or epidural injection; for low backache, epidural injection and perhaps spinal manipulation; for intercostal or cervical neuritis, paravertebral injection.

If one is looking for a specific that will cure all types of backache or any one of them, disappointment is certain. The application of heat, massage, systemic regulation, proper shoes and proper exercises are necessary after any minor surgical procedure.

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PNEUMOCOCCUS PNEUMONIA

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BOSTON

IMPORTANT advances have been made in the study of the pneumococcus. It was discovered in 1880, but that there are different types of pneumococci remained unknown for many years. Not until 1910 was it discovered by Neufeld and Haendel[1] that there were at least three fixed types. Their work was confirmed in this country by Dochez and Gillespie[2] in 1913. Between 1929 and 1932, Cooper et al,[3] working in New York, identified twenty-nine additional types.

A discovery was made by Neufeld[4] in 1902 the full value of which was not then recognized. He found that types of pneumococci could be identified when the organism in question was mixed with the homologous antipneumococcic serum through a swelling of the peripheral zone or capsule. The Neufeld method was adapted to the examination of sputum in 1932.[5] It constitutes an important advance in the recognition of types, and has proved to be simple, reliable and rapid.

The first use of specific serum — unconcentrated horse antiserum — in this country was made in 1913 by Cole and his associates[6] at the hospital of the Rockefeller Institute. By 1929 Cole[7] had treated 371 cases of Type 1 pneumococcus pneumonia, with the low mortality rate of 10.5 per cent, against an expected rate in untreated Type 1 cases of about 30 per cent. At the Massachusetts General Hospital, specific treatment was first employed in 1914, and has been in use since that time. The average amount for each patient in Cole's series was 420 cc. To give such a large amount entailed the use of complicated apparatus. It was therefore an important advance when in 1924 Felton[8] succeeded in concentrating anti-pneumococcic horse serum so that a small amount was potent.

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Others were unable to duplicate Cole's favorable results in Type 1 cases, and about fifteen years ago doubts were expressed as to the merits of the procedure. These doubts were resolved by a comparison of the results in serum-treated and control series in the large hospitals in New York and Boston; it was shown by these studies that the method was efficient, and particularly so when serum was used early in the disease. But up to 1931, since the specific treatment of pneumococcus pneumonia had been used for the most part only in the cities and the great medical centers, particularly in the East, and since many of the patients were often admitted late in the course of the disease, relatively few patients with pneumonia received the treatment.

Dr. George H. Bigelow, then Commissioner of Public Health in Massachusetts, appealed to the Commonwealth Fund, of New York, to finance a pneumonia program. This enterprise, carried out from 1931 to 1935 inclusive, proved highly successful and by the latest analysis of the case reports over 1700 patients with pneumococcus pneumonia have been treated with specific antiserum by nearly 1100 physicians.[9]

The example set by Massachusetts has been followed in other regions. In November, 1935, upstate New York inaugurated a similar program. New York City has recently done the same. New England is fairly well covered. Programs are in operation in New Jersey, Pennsylvania, District of Columbia, Maryland and Iowa, and are under consideration in many other parts of the country. Surgeon General Parran of the United States Public Health Service formed a Pneumonia Committee in the fall of 1937 to consider the matter from the point of view of the country as a whole, and to suggest measures to be taken with respect to the project.

Such programs as these are best planned as

community projects, and in most regions on a statewide basis. They require the co-operation of state departments of health and of the medical profession as a whole. In most communities it is desirable to have the project under the supervision of an advisory committee, including representatives from the state department of health, the medical profession and others interested. It should be administered by the state department of health, but must be actually carried on by the medical profession. It is well to appoint some member of the department of health who will collect and analyze the records, and answer questions as to details that will inevitably arise. There must be typing laboratories at strategic points in the community, manned by trained technicians. Their work must be maintained at a high standard, although this may be difficult in laboratories where only few specimens are sent in annually for typing. Supervision by the state bacteriologist may obviate this difficulty. In most communities it is advisable to obtain the serum by purchase rather than to manufacture it. It should be distributed freely to all who need it.

It is perhaps needless to say that pneumococcus pneumonia is a medical emergency, and that the first responsibility of the physician is to have the sputum examined to determine if the patient has a type of infection which is amenable to specific therapy.

We now realize that the earlier classification of pneumonia into lobar pneumonia and bronchopneumonia, so far as treatment is concerned, is of no practical importance, and that it is much more desirable to know the inciting agent than the pathologic anatomy. We have in consequence come to speak of pneumonia as due to Type 1 pneumococci, Type 2, and so on, regardless of whether it is lobar pneumonia or bronchopneumonia.

"Colds" in some way predispose to the development of pneumonia; just how, is not known. The investigations of Dochez and his associates[10] suggest that in some way the "cold" virus activates the pneumococcus, serves to disseminate it through the respiratory tract, and increases its virulence.

From the point of view of treatment it is necessary to realize that a large proportion of cases of pneumococcus pneumonia are due to types of pneumococcus against which there are specific antiserums. In pneumococcus lobar pneumonia, about 80 per cent of the cases are due to Types 1, 2, 3, 5, 7 and 8.[11] We have a specific therapy against all these types except Type 3. About sixty per cent of the cases of pneumococcus pneumonia (including both lobar pneumonia and bronchopneumonia) are due to these types.[12]

It is fortunately easy in most cases to recognize lobar pneumonia. It is often not difficult to recognize bronchopneumonia. In lobar pneumonia there is usually an explosive onset and a fairly uniform grouping of initial symptoms. Following or during a "cold" there occur a chill, pain in the side, cough, bloody sputum and elevation of temperature. It must be appreciated, however, that there are many atypical cases in the lobar pneumonia group, and we cannot expect all these symptoms to be present in all patients with lobar pneumonia. In bronchopneumonia certain of these initial symptoms drop out.

There are two conditions which are especially likely to be mistaken for lobar pneumonia. One is pulmonary infarction, and the other is acute pneumonic phthisis. With pulmonary infarction, there is a similar grouping of initial symptoms: pain, cough, bloody sputum and elevation of temperature. The bloody sputum, however, is likely to be pure blood mixed with mucus, whereas the sputum of pneumonia is typically rusty. In addition, if there is an opportunity to observe the course of the temperature we find that with infarction it rises more slowly than it does with lobar pneumonia. The circumstances under which the infarction occurs usually make the diagnosis fairly easy; that is, it usually follows trauma, operation or delivery. Acute pneumonic phthisis is fortunately rare. The symptoms of onset may resemble those of lobar pneumonia, but they are likely to be more atypical in grouping. The temperature is usually more irregular. The sputum is greenish rather than rusty. Also, tubercle bacilli are likely to be present. There is often a past history suggestive of tuberculosis. There is no leukocytosis.

As already noted, the first responsibility of the physician when he sees a patient with pneumonia is to determine the type of pneumococcus responsible for the infection. This cannot be told from the clinical aspects. The material on which a diagnosis of the type can be most readily made is the sputum. The sputum from the lower rather than the upper part of the respiratory tract is most likely to contain the inciting agent. No antiseptic should be added to the specimen, for it may be necessary to do a mouse inoculation and depend on the living organism for identification. The specimen should of course be sent at once to the laboratory.

It is important to take a blood culture in each case before the serum is given. A positive culture makes it possible to determine the type, if no sputum is obtained. Blood cultures are an indispensable guide to dosage.

In Massachusetts, the Department of Public Health now furnishes blood-culture material, so that there may be no obstacle to taking cultures

in all cases of pneumonia. Sputum can almost always be obtained for typing if the physician is insistent. There are, however, some cases where it cannot. Under such circumstances the diagnosis may be made from the blood culture, but this entails a loss of time. The determination of the type from the sputum by the Neufeld method may take only a few moments, that from blood culture may take eighteen hours or longer.

If sputum is unobtainable, it may be possible to determine the type of pneumococcus infection by means of a throat swab. Time does not permit a description of the method. Though it may lead to the rapid diagnosis of type, it often entails a considerable loss of time.

The Neufeld method is applicable to all material containing pneumococci. With patients who have a Type 1 or Type 2 pneumococcus infection, we may rely on the diagnosis of type and conclude that the pneumonic process is due to these particular organisms. This, however, is not true of the other types, and it is always desirable to repeat the typing in order to avoid the possible error of regarding an organism which inhabits the upper part of the respiratory tract as the cause of the pneumonic process.

It is important with respect to treatment to consider the factors which modify the outlook in pneumonia. One of the most significant of these is the age of the patient. The young are likely to recover and the old to die. Mortality rates in the teens are about 6 per cent with lobar pneumonia, and the rate rises to about 60 per cent at the age of sixty. The age of the patient should be taken into consideration in modification of the dosage, and larger doses should be given as patients advance in years.

Another vital factor in prognosis is the extent of pulmonary involvement. Patients in whom only one lobe is involved have a relatively low mortality rate, and those in whom several lobes are consolidated have a much higher rate. When one lobe is involved about 30 per cent of the patients die; where four lobes are involved, about 60 per cent. The extent of the involvement should modify the dosage.

It should be appreciated also that women who are pregnant or are in the postpartum period have a higher case-fatality rate than others, and this should modify the dosage.

Another essential factor is bacteremia. Patients with it have a mortality rate four to five times as great as those without it. It is therefore important to take a blood culture at once and others at suitable intervals throughout the illness, and to be guided by them with respect to dosage. The incidence of bacteremia increases with age.

There is also, surprisingly enough, a seasonal factor in prognosis. Analysis of the Massachusetts series showed that in 299 cases treated between April 1 and September 30 the mortality rate was 9.4 per cent, while in 527 cases treated between January 1 and March 31 the rate was about twice as high. The explanation of this is not entirely certain.

Another factor to be taken into consideration is the type of pneumococcus infection. Type 3 is the most serious. Unfortunately there is no effective serum against it. The mortality rate is a little over 50 per cent. Some time ago it was estimated that the rate in Type 1 was 25 per cent; analysis of a larger number of cases indicates that it is about 30 per cent. In Type 2 the rate is a little more than 40 per cent.

The results of specific serum therapy are shown both clinically and with respect to mortality rates. There is a favorable change in the clinical aspects of serum-treated cases, particularly when the treatment is begun early. If given very early, antiserum may actually abort the disease.

In general, the mortality rate in serum-treated cases has been reduced to about half what it would be without it. With Type 1 infection, a 5 per cent mortality or less may be expected in patients treated within the first twenty-four hours, and a 20 per cent mortality if they are treated within the first four days. The fatality rate rises with the lapse of time from the onset of the disease to the inauguration of specific treatment.

I shall not attempt to go into the details concerning the mortality rates under specific treatment, but I should like to emphasize the great value of its early application. Much depends upon the time at which the serum is given. The mortality rates with the other types against which we have serum — Types 2, 5, 7 and 8 — are not so well established as that for Type 1, but it is fair to say that the rates can be at least cut in half by the serum, and more if it is given early.

With respect to the Massachusetts series, it is clear that in Type 1 a 14 per cent mortality rate, in patients treated within the first four days, falls far short of the attainable goal. If the patients had been treated earlier in the course of the disease, and if more blood cultures had been taken, the results would have been better. Cultures were taken in less than half the cases. It is now appreciated that every patient should have a blood culture before the serum is given. It is realized also that the dosage in the Massachusetts series has been somewhat too low and should be increased.

The initial dose in Type 1 pneumococcus pneumonia should be at least 60,000 units, in Type 2,

Type 5 and Type 8 at least 100,000, and in Type 7 at least 60,000 or 90,000. The dose should be doubled if the patient is over forty, if treatment is begun after the first three days of the illness, if more than one lobe is involved, if the patient is pregnant or in the postpartum period, or if bacteremia is present. The first dose should not be given in one injection. The first is only a test injection and should not exceed 2 cc. in amount. After two hours, more serum should be given. This may well be the rest of the vial, and after two more hours a third injection, the contents of two vials, should be administered. If the volume per vial is 10 cc. and the potency 2000 units per cubic centimeter, 60,000 units will have been given. Further injections may be given at two-hour intervals to make up the desired number of units in the initial dosage decided upon. No single injection, however, should exceed 50 cc. Additional injections of 40,000 to 60,000 units should be given every three to four hours in all severe cases, and in any case in which the temperature response is not favorable, in which bacteremia is present, regardless of the clinical course, or in which there is a spreading lesion.

Time does not permit a consideration of the precautions to be exercised in the intravenous use of alien serum. Physicians should be familiar with the contraindications before the serum is given.

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Discussion

Dr. Sven M. Gundersen, Hanover: We have been very fortunate indeed in having Dr. Lord come from Boston to speak to us about this extremely important problem.

The Pneumonia Service in New Hampshire was begun four months ago. The problem has been rather difficult. In the first place, we have been very much restricted as to funds. Those available have come from the United States Public Health Service, and even now we find that we are running short. There have been special difficulties, too, because we are dealing with a rather small, scattered population, and it has been hard to supply adequate typing service for all parts of the State. So far, the typing service has not been adequate, but we hope that it will be improved in the fall.

To my knowledge, there have been more than 85 cases of pneumonia treated with serum supplied by the State. In 56 of these I have received reports from the doctors concerned. Six had a fatal termination. Careful review of these fatal cases makes it seem probable that only 2 deaths can be attributed to failure of the serum treatment. In the other 4 cases there were serious complicating diseases, and in some of these, serum treatment was not instituted early enough and the serum was not given in adequate dosage. Fifty patients have recovered, and most of them have been Type 1 cases.

As most of you are aware we are now supplying serum for Types 1, 2, 5 and 7. We are also supplying blood-culture outfits for every patient whose sputum is typed in one of the two accepted laboratories.

I should like to make a plea for the conscientious return of reports on serum-treated cases, because only thus can we find out what we are accomplishing.

Dr. Francis H. Nolin, Claremont: Dr. Lord has pointed out the value of using serum in the treatment of pneumonia. He has also told us about the work that has been carried out by the Massachusetts Pneumonia Commission, as well as the correct methods of diagnosing the different types of pneumonia. We physicians in New Hampshire are most fortunate to hear Dr. Lord speak on this subject, especially at this time, since the State Board of Health this year introduced a pneumonia control program.

At present we have only two laboratories equipped to do pneumonia typing, one in Hanover and the other in Concord. Sputum can be sent to either laboratory, and an early diagnosis and report can be given. It is hoped that at a later date we may have many more approved typing stations throughout the State, thus offering every community the advantages of early diagnosis, especially in the cases that are found to be of a type where serum can be used. It is also hoped that we shall have available adequate funds to build up an educational program to enlighten the people in this method of treatment, as well as to instruct physicians in the use of antipneumococcus serum. If this is done, there should be a marked decrease in the mortality rates in these cases over a period of years.

We know that in the treatment of pneumonia very large doses of serum are necessary, and we hope to have adequate amounts of serum for Types 1, 2, 5, 7 and 8, and possibly later on for some of the other types, as serums for them are developed on a commercial basis. The use of serum should be encouraged, and we in New Hampshire ought to bring about a marked decrease in the mortality rate, comparing favorably with the results attained in Massachusetts and other states.

Dr. Emery M. Fitch, Claremont: In view of the value of serum treatment, and of the number of commercial vaccines on the market today, I should like to ask Dr. Lord if there is any vaccine against pneumonia which is efficacious.

Dr. Arthur W. Burnham, Lebanon: Dr. Gundersen spoke of funds having been made available for the Pneu-

monia Service in New Hampshire. We can all do considerable missionary work in our own communities by spreading knowledge of this forward step in the control of pneumonia, to my mind the greatest advance in biological medicine since the discovery of insulin. We should be rendering a public service if we exerted every possible influence upon legislators and others in public office to see that funds are made available for this work. We have seen many comparatively young men, the breadwinners of their families, stricken by pneumonia. There is a distinct economic loss that we are continually suffering from this disease, and this is a point that we can well bring home to the laity and to our legislators.

While my own practical experience has been limited, I have been fortunate in being close to Hanover, where I could easily get the services of a typing station, and where I could also call Dr. Gundersen by telephone for consultation. During the last two or three months in my little town of Lebanon, I know of 6 cases that were treated with 100 per cent recovery. I had 3 cases, 2 of which were Type 7 and 1 was Type 5. One patient had a positive blood culture; all 3 had a crisis and a rapid and uneventful convalescence.

DR. LORD (closing): As to the use of vaccines, there are two aspects of the problem. One is the question whether a vaccine will prevent pneumonia. The other is the question whether it is of value in the treatment of pneumonia.

In laboratory animals, an otherwise fatal pneumococcus infection can be prevented by previous vaccination of the animal with the homologous organism. There are underway in the CCC camps experiments, inaugurated by Felton, which give promise of the prevention of pneumonia by vaccination. The results are not yet available.

Given an outbreak in a small community, such as a school or institution, it would be justifiable to vaccinate the individuals in that community with the organisms concerned in the epidemic in the hope of stopping it. Thus far, we cannot say that vaccination is a practical procedure against pneumonia for the general population.

Some physicians are using vaccine in the treatment of an existing pneumonia. There is, however, no justification for such use. It is difficult to understand how a vaccine, which is nothing but a suspension of dead organisms, can accomplish as much immunization of the individual as the infection itself. The belief has been that one might accelerate this immunization by the use of dead organisms early in the course of the disease. This is pure theory. From a laboratory point of view there is no evidence that one can speed up the production of antibodies in an already infected individual. There is no evidence of the value of vaccine in the treatment of pneumonia; there is of course a difference between vaccine and serum, one being a suspension of the organisms and the other serum from immunized animals.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24391

PRESENTATION OF CASE

First Admission. A twenty-six-year-old, white, American truck driver entered the hospital with the complaints of headache of four weeks' duration and abdominal distention and peripheral edema of three weeks' duration.

Six weeks before entry he had a head cold and slight sore throat accompanied by a cough productive of thick white sputum. Two weeks later he began to have severe, lancinating frontal and periorbital headaches which interfered with his sleeping. These were somewhat relieved by Anacin and became somewhat less severe during the week before entry. Three weeks before entry he noticed that his ankles swelled when he was sitting around the house. The swelling subsided when he walked. About that time he also began to notice epigastric discomfort and fullness after meals. This gradually increased to persistent abdominal fullness with epigastric distress and vomiting after each meal. He had no chills or fever, no chest pain or hemoptysis, and no hematemesis, diarrhea or abnormal stools. He had lost 10 to 15 lb. during the month before entry.

At the age of four he had had rheumatic fever and remained in bed for a year. He had pain in his ankles and knees at that time, and during each winter up to the age of fifteen he had recurrence of ankle and knee pain lasting for two or three weeks. He also had frequent upper respiratory infections during the winter months. Since childhood his activity had been slightly limited by exertional dyspnea and palpitation, but he had always been able to do hard manual work. He had no nocturnal dyspnea. At one time he was told he had a leaking heart valve. His family history was negative.

Physical examination revealed a well-developed, fairly well-nourished man lying flat in bed in no obvious discomfort. There was a questionable icteric tint to the sclerae. The apex impulse of the heart could be felt in the sixth interspace in the anterior axillary line. At the apex there was a systolic murmur and a loud blowing diastolic murmur with a diastolic thrill. There was also a blowing diastolic murmur heard down the left border of the sternum, and a systolic murmur with a faint thrill at the base. P_2 was snapping and louder than A_2. The blood pressure was 140 systolic, 70 diastolic. The lungs were clear. The liver was tender and could be palpated 8 cm. below the costal margin. It seemed to show definite pulsation. There was slight shifting dullness in the abdomen, but no peripheral edema.

The temperature was 98.6°F., the pulse 90. The respirations were 20.

The urine had a specific gravity of 1.020 and contained 4+ albumin with a few red cells in the sediment. The blood showed a red-cell count of 5,200,000 with 70 per cent hemoglobin and a white-cell count of 8600 with 76 per cent polymorphonuclears. The guaiac test on the stool was twice negative and on the vomitus 2+. The blood Hinton test was negative. The nonprotein nitrogen of the blood was 29 mg. per cent, the chlorides were equivalent to 104 cc. of N/10 sodium chloride and the van den Bergh was slightly above normal. The sedimentation rate was 0.2 mm. per minute. An electrocardiogram showed moderate right-axis deviation and wide notched P waves. The P-R interval was 0.19 sec.

An x-ray of the chest showed marked enlargement of the heart to both sides. The left border was fairly straight. Both costophrenic angles were obliterated, and the lung markings were very prominent. An x-ray of the abdomen showed no definite enlargement of the spleen or liver. Both organs were poorly outlined. There was no evidence of fluid in the abdomen.

He was digitalized and showed very rapid improvement. At the end of five days all vomiting had ceased, the liver was much smaller, and the urine had become normal. However, he continued to have headaches, and during the first ten days these became increasingly severe. A lumbar puncture was done on the eleventh day which showed normal dynamics. However, the spinal fluid was slightly cloudy and contained 64 lymphocytes and 5 monocytes per cubic millimeter. The total protein was 153 mg., the sugar 31 mg., and the chlorides 643 mg. per cent. The spinal fluid Wassermann was negative, and a culture yielded 10 to 12 colonies of yeast. The lumbar puncture gave some relief from headache. A neurological examination two days later was entirely negative except for stiffness of the neck and a positive Kernig sign. A second lumbar puncture on the sixteenth day again showed normal dynamics. The fluid contained 400 mononuclear cells; the protein was 148 mg., the sugar 30 mg. and the chlorides 603 mg. per cent. The goldsol curve was 0001110000. The blood sugar taken at the same time as the lumbar puncture was 65 mg. per cent.

A culture of the spinal fluid gave no growth on blood or Sabouraud's medium.

About that time his temperature began to rise daily to 100°F. and it continued to do this until he was discharged nine weeks later. Also, the white-cell count of his blood rose to 13,000 with 71 per cent polymorphonuclears and from then until discharge fluctuated between 10,000 and 20,000. No abnormalities were noted in the blood smears. The red-cell count temporarily fell to 4,000,000, but returned to 4,800,000 before discharge. Seven blood cultures gave no growth, and five tuberculin tests using dilutions varying between 1:20,000 and 1:100 were all negative. Four more electrocardiograms were taken and showed no significant changes. In two of them the P-R interval was 0.20 sec., and they all showed T-wave abnormalities consistent with a digitalis effect.

An x-ray of the chest on the eighteenth day showed clearing of the hazy areas and decrease in the size of the heart. Skull x-rays were negative. The headaches gradually diminished in frequency, and on the twenty-ninth day the neurological examination was entirely negative. One week later the blood sugar was 76 mg. per cent, and the following day a third lumbar puncture showed an initial pressure of 110 mm. of water, with clear and slightly xanthochromic fluid. It contained 350 lymphocytes, 20 large mononuclear cells and no red cells. The total protein was 129 mg., the chlorides 664 mg., and the sugar 23 mg. per cent. A blood sugar taken at the same time was 117 mg. per cent. The goldsol curve was 4455454321. Two days later the sedimentation rate was 0.2 mm. per minute. One week later in the middle of the seventh hospital week he vomited once or twice, but his neurological examination continued to be negative. However, there had been a definite change in the systolic murmur at the base of the heart. It was rougher and was transmitted into the neck where a thrill could be easily felt. Otherwise the physical examination was essentially unchanged. No petechiae had appeared, and the spleen could not be felt. A lumbar puncture at the end of the seventh week showed an initial pressure of 230 with a cell count in the fluid similar to that of the previous one. The protein was 166 mg., the sugar 23 mg. and the chlorides 595 mg. per cent. The goldsol curve was 0014554321. Guinea-pig inoculation of the fluid failed to produce tuberculosis in the animals. A neurological examination nine days later was again entirely negative. A lumbar puncture at the end of the ninth week showed similar spinal-fluid findings as before, with a sugar of 17 mg. per cent and a 4455555553 goldsol curve. His intermittent headache and vomiting had gradually diminished, and he was finally discharged in the middle of the eleventh week.

Final Admission (three months later). Shortly after discharge his headaches entirely disappeared. He remained quite comfortable leading a bed and chair existence until three weeks before re-entry, when he developed a head cold with slight sore throat. He had continued to have some dyspnea on exertion, but at about the onset of the cold he had for the first time an attack of nocturnal dyspnea which awakened him from sleep. The attack was relieved by sitting up, and he was able to continue sleeping flat in bed. After that, however, his appetite was very poor and he had generalized malaise with epigastric distress and abdominal fullness. He had inconstant nausea and occasional vomiting. One week before re-entry he began to cough up blood-streaked, mucoid sputum and had had pleuritic chest pain in the right axilla. On the day of re-entry he noticed edema of the ankles.

Physical examination revealed a slightly icteric, chronically ill, undernourished, slightly dyspneic man lying propped up in bed. The heart was grossly enlarged. The rate was 140, and the rhythm was totally irregular, with a pulse deficit. There was a frank systolic murmur over the whole precordium, transmitted into the neck, and there was a blowing aortic diastolic murmur. The apical diastolic murmur was not heard. The blood pressure was 140 systolic, 40 diastolic. The lungs showed dullness with diminished breath sounds and a few rales at the right base. The liver edge could be palpated 5 cm. below the costal margin and was definitely tender and pulsating. The spleen could not be felt, and there was no significant amount of fluid in the abdomen. There was slight edema of the ankles.

The temperature was 97°F., the pulse 140. The respirations were 25.

The urine contained a trace of albumin and occasional red cells and casts. The blood showed a red-cell count of 4,700,000 with 90 per cent hemoglobin and a white-cell count of 18,000 with 75 per cent polymorphonuclears. An x-ray of the chest showed dullness at the bases.

He died on the third hospital day.

Differential Diagnosis

Dr. Paul D. White: I can say at the beginning that this combination of headaches with abdominal distention and peripheral edema is very unusual. My first thought is that he had a sinusitis complicating a respiratory infection. There may have been an increase in intracranial pressure due to infection or a local lesion, but we have as yet no proof of either.

Just a word about the decrease of swelling of the ankles on walking. That may occur no matter what the cause of the edema. Standing still will favor the occurrence of swelling of the feet

from any cause. Movement of the legs will tend to decrease the swelling unless the exercise is extreme; that is, mild to moderate exercise favors the circulation, standing still does not. We note this in patients with chronic constrictive pericarditis, varicose veins, and even mild heart failure.

One must think of various causes for abdominal enlargement and swelling of the legs precipitated by respiratory infection — heart failure, pericarditis, nephritis. Heart failure is the most likely in the absence of fever or evidence of nephritis. Moreover, the edema was not generalized but dependent.

In this patient's past history a respiratory infection doubtless preceded each attack of rheumatic fever, year after year.

It is almost certain that he must have had an abnormal heart after so much rheumatic infection. There was undoubtedly some limitation of his activity and ability to carry on vigorous effort, but he did not have enough heart failure to prevent his lying flat in bed.

The description of the diastolic murmur is not characteristic of mitral stenosis, but the thrill accompanying it certainly would favor such a diagnosis. It is possible that the aortic diastolic murmur that he also showed was transmitted to the apex and may have been part of what was heard in diastole; in other words, at the apex one usually hears superimposed the two diastolic murmurs of aortic regurgitation and of mitral stenosis, which may explain the description of the murmur given here.

"There was also a blowing diastolic murmur heard down the left border of the sternum." That is surely indicative of aortic regurgitation. It does not say where the systolic thrill was felt.

DR. A. THORNTON SCOTT: Over the aortic area.

DR. WHITE: That would support the interpretation of aortic valve disease accompanied by mitral disease, that is, of aortic stenosis and regurgitation and mitral stenosis and regurgitation.

He had a fair pulse pressure, but not indicative of a high degree of aortic regurgitation; there may have been enough stenosis to limit the aortic regurgitation. The fact that the liver showed pulsation makes one also wonder if tricuspid stenosis was present. The liver may pulsate in the absence of tricuspid stenosis when there is a high degree of congestive failure. Functional tricuspid regurgitation, with heart failure and engorgement of the liver, may be accompanied by pulsation of the liver. The most marked liver pulsations I have encountered have, however, been in patients with tricuspid valve stenosis. There is no note here of specific murmurs in the tricuspid valve region. Sometimes one does find localized systolic and mid-diastolic murmurs at the lower end of the sternum, but cases are rare in which the degree of stenosis is enough to produce murmurs. In order to judge about the liver, that is, the question of congestion of the liver, one would like to have had an observation of the neck veins. There is none noted here.

DR. CHARLES R. ALLISON: There was pulsation in the neck veins.

DR. WHITE: That is confirmatory of congestive heart failure's being responsible for the large liver.

There is no note about the heart rhythm. I judge that it was normal. This is a rather important point in the interpretation of the diagnosis later. The temperature was normal. The pulse was somewhat elevated. The urine showed a fairly normal specific gravity with albumin and a few red cells, which would be consistent with congestive failure. There was no anemia or leukocytosis.

The first electrocardiogram is almost absolutely characteristic of mitral stenosis, not of congenital or pulmonary heart disease, which might also cause abnormal right-axis deviation. The second electrocardiogram shows a change in the T waves probably resulting from digitalis. The P-R interval was 0.19 sec., which would be prolonged for a child, but at this patient's age it is just within normal limits and may or may not mean a slight delay in conduction.

The x-rays show the heart to be enlarged and of a characteristic shape. As you see, the first one taken is consistent with rheumatic heart disease with mitral valve involvement. The straight left border is such as one sees frequently in mitral valve stenosis, with prominence of the right ventricle and pulmonary artery. The left auricle does not show distinctly, although the hilus shadows are somewhat displaced outward. The shape of the shadow is not that of a pericardial effusion and we already know from physical examination that the heart is large, the apex being felt in the anterior axillary line in the sixth space.

"He was digitalized and showed very rapid improvement"; this indicates, to my mind, cause and effect. It is probable that the digitalis did have a favorable effect and that, therefore, congestive heart failure was the chief factor responsible at this time for most of his symptoms, without the need of implicating any serious infection. We also note that his temperature had been normal and that he had had no chills. Almost all his symptoms could be due to congestive failure and would clear up with digitalis.

"However, he continued to have headaches and during the first ten days these became increasingly severe." There is the rub! The headaches were severe, even while he was improving so far as all

other findings were concerned, including the liver enlargement and the gastric irritation.

Were the eyegrounds examined? There is no note of that. They were probably normal.

I asked Dr. Kubik the significance of the findings in the spinal fluid. The total protein was much increased; the normal is about 50 mg. The sugar was much decreased; the normal is about 85 mg. The chlorides were also decreased to a considerable extent, being normally about 700 mg. These findings suggest infection rather than a simple irritation such as aseptic meningeal irritation.

DR. TRACY B. MALLORY: I find a statement that the fundi were within normal limits but the margins were not so clear as they were previously.

DR. WHITE: One wonders whether the positive culture of the spinal fluid was the result of a contamination. Later they used Sabouraud's medium in a search for yeast and failed to find any organisms.

The history now states that the temperature began to rise and that some fever continued until he left the hospital, nine weeks later. In other words, during nine of the eleven weeks he had fever to about 100°F. daily. On looking at the chart we find that it was not a septic temperature by any means. It went up to 100 and then down to 99°F. by mouth, but at the end it was down to normal. This is not at all the temperature chart that we should expect in the usual case of subacute bacterial endocarditis, although there may be great variations; infrequently there are afebrile periods in the so-called abacterial stages.

Repeated blood cultures showed no growth and, considering the care with which blood cultures are taken nowadays, that fact militates against the diagnosis of bacterial endocarditis unless it were on the right side of the heart, which is very unlikely here since the chronic valvular disease is on the left side.

The electrocardiogram taken then shows a little increase in the P-R interval. The difference is so slight that one should not pay too much attention to it. There is, however, a change in the T waves between the first and later electrocardiograms. After the first they became inverted, which is consistent with a digitalis effect. They are not "pericardial disease" electrocardiograms. In pericarditis we usually have a striking change in the T waves in all leads.

X-ray films taken at that time show clear lung fields. The heart shadow looks slightly smaller, but there is still congestion in the lungs.

I suppose a serious sinusitis would be detected by the x-rays of the skull; that possibility, the diagnosis of sinusitis, which we considered originally as an explanation of the headaches, is pretty much ruled out by the x-rays.

Whatever he had in his head apparently cleared up spontaneously, which would not be typical of neoplasm or serious infection. Recovery might occur with a local lesion, such as an infarct, or perhaps from meningeal irritation secondary to some infection in the skull itself, such as sinusitis.

The xanthochromic fluid suggests that there may have been some bleeding; there is no evidence that it was produced by previous lumbar puncture. However, the fluid was only slightly xanthochromic, which, as Dr. Kubik told me, is more in keeping with a high protein content.

"However, there had been a definite change in the systolic murmur at the base of the heart." Can anyone here say whether it was a striking change?

DR. SCOTT: I think it was.

DR. WHITE: It may have been the result of improvement in his circulation. Aortic stenosis is frequently or occasionally missed because of the occurrence of heart failure, and after improvement of the circulation, thrill and murmur are found and they become more marked with recovery. So we have to balance that against the possibility of the development of vegetations on the aortic valve, which in turn might have been responsible for a cerebral infarct. We could have an increase in systolic murmur and thrill with the superimposition of large vegetations on the aortic valve, but we should hardly expect him to improve as much as he had if that were the story.

Was he having a fever at the time of discharge?

DR. MALLORY: It was down to 99°F. (rectal).

DR. WHITE: I wonder if he had fever at home between the time of his discharge and his return a few months later. We have no note of that. It seems possible that he continued to have a low-grade febrile reaction, but he was quite comfortable at home for more than two months after his discharge and until three weeks before re-entry. He died three days after re-entry to the hospital. He developed a head cold and slight sore throat, a clear cause for an exacerbation of his disease, whether heart failure or rheumatic infection.

In this final history of the case, the patient showed more pulmonary involvement than failure of the right ventricle; in other words, the course suggests left ventricular failure. On the other hand, nocturnal dyspnea may be due to other things, such as pulmonary infarction.

Apparently there was auricular fibrillation with the same degree of right-axis deviation and a rapid pulse—a terminal arrhythmia which may de-

velop even in the presence of bacterial endocarditis. If auricular fibrillation had been present earlier it would have been a point against bacterial endocarditis. Here the aortic murmurs were preponderant; the mitral diastolic murmur was not heard. Some of the apical systolic murmur was probably mitral in origin, however. There was an increase in pulse pressure which may mean more aortic regurgitation. It was in the right axilla that he had the chest pain at the time he coughed up blood.

The same question comes up again as to whether the liver enlargement and pulsation indicate heart failure or tricuspid valve disease. The liver had returned to normal size before, so it does not seem likely that there was much tricuspid stenosis. There was no note of clubbing of the fingers at any time. I suppose none was found or it would have been indicated. Apparently the spleen could never be palpated.

There are certain diagnoses we can make without much hesitation and others that we must question. Rheumatic heart disease with involvement of mitral and aortic valves must be considered quite certain. Terminal auricular fibrillation, congestive heart failure, and acute respiratory infection precipitating failure are other diagnoses that can be made with certainty. Pulmonary infarction is almost certain. There is a question of tricuspid valve involvement with stenosis.

What was the cause of the headaches? What was the cause of the fever? Those are the two chief problems. Did he have a cerebral infarction which caused the headache and the changes in the spinal fluid, with later recovery from the infarction such as we would expect to be associated with the disappearance of the headache and improvement of the spinal-fluid findings? Infarct coming as a result of embolism from the heart might be either the result of the breaking off of a bit of thrombus in the left auricle without bacterial endocarditis or, more readily, a sequela of bacterial endocarditis. Cerebral infarction does not prove the presence of bacterial endocarditis. It may simply mean rheumatic heart disease with mitral stenosis. It seems much less likely that a sinusitis or other skull infection could have caused such prolonged symptoms and the abnormal spinal-fluid findings. We have no clear indication of brain abscess or of a serious meningitis, tuberculous or septic; an aseptic meningitis cannot be ruled out. The cerebral infarction, if present, must have been in the frontal lobe or other relatively "silent" area for there was no localization from the neurological standpoint: no weakness of one side of the body, hemiplegia or monoplegia. As the cause of the headaches and meningeal irritation, I should rather favor cerebral infarction than other diagnoses although the onset apparently was not abrupt. We have no clear indication as to whether the headache started suddenly. The history simply says that two weeks after a head cold he had severe, lancinating frontal and periorbital headaches.

Rheumatic infection can be stirred up easily by respiratory infections and would explain most of the clinical picture so far as the temperature reaction is concerned, and I should rather favor the diagnosis of acute rheumatic infection, probably continued right through the illness, perhaps recurrent after the second cold, over the next best diagnosis, that of subacute bacterial endocarditis; there is, however, not much to choose between them. The congestive failure and then the heart infection were apparently set off by the respiratory infection at the very beginning. There may have been some other unusual infection or lesion that I cannot diagnose.

Dr. Walter Bauer: We raised the question whether he had yeast meningitis, because someone found yeast-like organisms in the first spinal fluid examined. This finding was never confirmed. The other question frequently asked was, Is this tuberculous meningitis? I thought that the spinal-fluid findings were more those of an aseptic meningitis accompanying a subacute bacterial endocarditis than those of tuberculous meningitis.

Dr. Edward F. Bland: I saw the patient about midway during the first admission after the clear spinal fluid was obtained. I did not see him during the last episode. The question which we were asked to express an opinion on was whether active rheumatic fever could explain the spinal-fluid findings. I considered them at some length and wrote this: "I do not believe we can explain them directly on the basis of rheumatic fever. In the patients at the House of the Good Samaritan we have seen only one instance, other than embolism or chorea, with cerebral manifestations complicating severe rheumatic fever; namely, thrombosis of a lateral sinus without further demonstrable cause other than rheumatic infection and congestive failure." The findings in that case were not comparable to this. Finally, I wrote: "I believe this patient had an active process in his heart. The clinical course favors rheumatic fever, but subacute bacterial endocarditis cannot yet be excluded. Until proved otherwise, it seems to me more likely that the spinal-fluid findings and cerebral complications are secondary to embolism rather than to an unrelated disease process."

DR. MALLORY: Have you any suggestions, Dr. Jones?

DR. T. DUCKETT JONES: I regret that the pathological findings are known to me so I cannot discuss the case without prejudice. The clear reasoning of Dr. White seems logical and covers the situation adequately from a clinical point of view. The history and physical findings suggest strongly to me that careful histological search would reveal the presence of active rheumatic changes in the heart, despite what other findings are present. This case illustrates the value of trying to explain by a single diagnosis the major features of a given diagnostic problem. Rheumatic fever will not give the spinal-fluid changes found in this patient. It would be wise to consider seriously subacute bacterial endocarditis in any patient with rheumatic heart disease and severe headaches.

DR. JAMES B. AYER: I saw this patient once or twice. Since I had previously seen fluids of this character in undoubted cases of septic embolus, I felt that the neurological picture and the spinal fluid picture were compatible; but as I watched the sugar and the chlorides go down, I leaned more to some chronic infection such as tuberculosis, and I was temporarily quite relieved and satisfied when someone said there was a yeast present, and it was probably a yeast meningitis. That was subsequently disproved, however, so I should agree that this whole array of fluids is not quite typical of subacute bacterial endocarditis; but some endocarditis cases, especially in the early stage, are atypical.

CLINICAL DIAGNOSES

Rheumatic heart disease with mitral and aortic stenosis and insufficiency.
Tricuspid disease?
Auricular fibrillation.

DR. WHITE'S DIAGNOSES

Rheumatic heart disease.
Mitral and aortic stenosis and regurgitation.
Tricuspid valve disease?
Auricular fibrillation.
Congestive heart failure.
Pulmonary infarction.
Acute rheumatic infection or subacute bacterial endocarditis; more likely the former.
Cerebral infarction or aseptic meningeal irritation; more likely the former.

ANATOMICAL DIAGNOSES

Rheumatic heart disease with aortic, mitral and tricuspid stenosis.
Verrucous endocarditis, aortic and mitral valves; ? "nonbacterial phase" of subacute bacterial endocarditis.
Pulmonary infarcts.
Renal infarct.
Cerebellar infarct, minute.
Meningitis, chronic, slight.

PATHOLOGICAL DISCUSSION

DR. MALLORY: When we have a case as thoroughly worked up as this and are nevertheless unable to make a clinical diagnosis it very often turns out that the autopsy does not answer the question either. We have to admit that here. The man had very obvious old rheumatic heart disease with severe involvement of the aortic and mitral valves and some involvement of the tricuspid, but not enough to indicate functional impairment. The chordae tendineae were thickened, but the valve margins were not retracted and there was not much stenosis. The rest of the viscera were essentially negative except the lungs, which showed multiple infarcts. We found a normal-sized spleen, and normal-sized kidneys. One of the latter contained a gross infarct, but no others were seen microscopically in either the spleen or the kidneys.

The heart showed in addition to the old lesions, some fresh vegetations which were of fairly good size. Grossly they were quite suggestive of bacterial endocarditis. We sacrificed these vegetations to bacterial culture, ground them up, and then were unable to grow anything; so unfortunately we have no histologic examination of the vegetations. In the myocardium itself, we have not been able to find anything that suggests rheumatic infection. There was a myocardial degeneration of the lipoid type which we often see in patients with very severe anemia and seldom with other conditions, but it is not, so far as I am aware, common with rheumatic fever. Dr. Kubik will tell us about the brain. We could quickly rule out yeast meningitis.

DR. CHARLES S. KUBIK: There was one very small infarct which could be seen with the naked eye, but which probably would not have caused the symptoms. Microscopic sections showed a patchy chronic inflammatory reaction in the subarachnoid space, either a resolving or a chronic, progressive process. No yeast organisms were seen, and tuberculosis can almost certainly be ruled out. The findings are similar to those we have observed in other cases of subacute bacterial endocarditis with symptoms of meningitis, and I believe that is the diagnosis here. The meningeal reaction presumably results from emboli arrested in small meningeal arteries or arterioles.

DR. BAUER: Were not the findings in one or two of the aseptic meningitis cases something like this?

DR. KUBIK: Yes, there has been a lowering of the chlorides in some of them. We have had no postmortem examinations. I do not believe there have been any deaths.

DR. BAUER: What was your final diagnosis, Dr. Mallory?

DR. MALLORY: I thought it was bacterial endocarditis, but I have no proof of it.

DR. JONES: Do you think that the absence of positive blood cultures during life and of positive cultures of the valve tissues is against the diagnosis of subacute bacterial endocarditis?

DR. MALLORY: Undoubtedly. On the other hand, it is very unfortunate that we did not directly stain these vegetations because bacteria in such lesions can sometimes be stained when they cannot be grown.

DR. JONES: In a certain number of typical bacterial endocarditis cases no bacteria are distinguishable in the microscopic sections of the involved valves. In these instances clumps of granular masses are found in the thrombus at the valve surface. These granules are readily stained but definite organisms cannot be made out. But negative blood cultures do not necessarily exclude a diagnosis of bacterial endocarditis. The final proof of the presence of the disease is dependent upon microscopical findings in the valves involved.

DR. BAUER: He was not in the hospital during much of his illness and could perfectly well be in a bacteria-free stage when he was here and have died of heart failure. I do not see why we should be disturbed by the negative blood cultures.

DR. JONES: That is what I meant to intimate.

DR. WHITE: He died of heart failure rather than of the infection.

DR. MALLORY: If we say that it is not bacterial endocarditis, we have to assume that he had an aseptic meningeal infection of some sort, and we must admit we have not the remotest idea of its etiology.

A PHYSICIAN: Where were the fresh vegetations?

DR. MALLORY: On both the aortic and mitral valves.

A PHYSICIAN: Were they large?

DR. MALLORY: Yes; they were a little larger than we would expect to see with an acute rheumatic process.

* * *

DR. WHITE (later note): This is the first case of subacute bacterial endocarditis, as seems probable on full analysis, that I have known to have been complicated by a meningitis; the cases seen in this hospital that have shown more clear proof should be reported in the literature, which as yet has little to say on the subject.

Blumer[1] in his monograph on subacute bacterial endocarditis stated: "Symptoms referable to the nervous system are of frequent occurrence in subacute bacterial endocarditis. *Headache* is the most common symptom. It may occur early as a result of the toxemia or may not appear until the embolic period when it may be associated with cerebral embolism or rarely with meningitis." Blumer goes on to cite a case reported by Claude of a boy of eighteen who showed marked meningeal symptoms and an abnormal spinal fluid, and at autopsy, slight meningitis of the cerebral cortex. A report by R. N. DeJong,[2] discussing central-nervous-system complications in subacute bacterial endocarditis seen at the University Hospital in Ann Arbor during a period of five years, states that the central nervous system was examined in 13 necropsies of cases with symptoms suggesting involvement of the central nervous system or meninges. In all 13 cases pathologic changes were found in the brain or meninges: in 6 of these there were emboli or infarcts secondary to emboli; in 4 there was purulent metastatic meningoencephalitis on the basis of multiple small abscesses in the brain and in the meninges; and 3 showed simply edema, congestion, and hyperemia of the brain without more specific changes.

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CASE 24392

PRESENTATION OF CASE

A thirty-six-year-old, white, native-born farmer entered the hospital with a complaint of abdominal pain of two months' duration.

The patient had always been in excellent health until two months before entry, when he began to have attacks of dull, not severe, pain in the right lower quadrant, which came on ten or fifteen minutes after breakfast and occasionally after lunch, and lasted two or three minutes. At the same time the right testicle became swollen and heavy without any pain or tenderness or accompanying urinary symptoms. The bowel movements, which had always been regular, had remained unaffected and were in no way related to the pain. The character of the pain remained relatively unchanged during his illness although during the two weeks before entry it occasionally occurred at night. During this period of two weeks he also had nocturia twice each night. He noticed that he became more easily fatigued

and stated that he had lost 8 lb. during his illness. He never had any bloody or tarry stools, diarrhea, nausea, vomiting, jaundice or other gastrointestinal symptoms. He continued to have a good appetite.

His past and family histories were noncontributory.

Physical examination revealed a well-developed, well-nourished, somewhat pallid man lying quietly in bed in no discomfort. The heart and lungs were negative, and the blood pressure was 130 systolic, 70 diastolic. In the right lower quadrant just above Poupart's ligament there was a firm, non-movable, slightly tender, smooth mass measuring 8 by 3 cm. The liver edge was sharp and palpable just at the costal margin. The tip of the spleen could be felt just under the left costal margin. There was no abdominal spasm. On rectal examination the prostate was found to be smooth and uniformly hypertrophied. Just beyond the right lobe of the prostate beneath the mucosa of the bowel and either in its wall or external to it, was a firm, movable, non-tender, bean-sized mass. The right scrotal sac was larger than the left, fluctuant, and non-tender. It transilluminated well, and the venous plexus was enlarged and tortuous.

The urine examination was negative. The blood showed a red-cell count of 2,980,000 with 55 per cent hemoglobin. The white-cell count was 21,000 with 70 per cent polymorphonuclears. The smear was negative except for moderate achromia. The stools gave a 4+ guaiac test on two occasions. A blood Hinton was negative, and the serum protein was 6.1 gm. per cent. A gastric analysis showed 0 units of free and 6 units of combined fasting acid, 8 units of free and 12 units of combined acid after alcohol, and 10 units of free and 16 units of combined acid after histamine. The gastric contents were guaiac negative. A tuberculin test using 1:20,000 dilution was negative.

An x-ray of the chest was negative. A barium enema flowed to the cecum and through the ileocecal valve without delay. The cecum appeared slightly spastic but otherwise showed no abnormalities. The ileum, after emptying, appeared wider and contained more barium than normal. On the ninth day a gastrointestinal series showed thickening of the gastric rugae, particularly in the antrum. At the end of six hours there was a small residue in the stomach, the remainder of the meal lying in the terminal ileum and colon and extending as far as the hepatic flexure. The cecum was poorly filled. A repeat barium enema confirmed the previous finding of a spastic cecum, which was difficult to visualize adequately. When the barium reached the region of the ileocecal valve, it passed out readily into the small bowel but failed to fill the cecal tip. A definite mass was palpable corresponding to this region, but it could not be determined whether this mass arose in the cecum or was extrinsic to it and produced sufficient pressure on the cecum to render its adequate visualization impossible.

A laparotomy was performed on the thirteenth day.

Differential Diagnosis

Dr. Claude E. Welch: The etiology of right lower-quadrant masses is always of great interest. In youth, they nearly always represent appendiceal abscesses; in adults, carcinomas of the cecum are nearly as common. A few more bizarre types must be considered in either case.

In this patient we have a young adult who, after initially trivial symptoms, finally becomes ill enough in two months to go to the hospital. Despite the meager symptomatology, examination reveals several interesting features. There is a large right lower-quadrant mass obviously pressing on the spermatic vessels. There is a second nodule palpable by rectum, strongly suggestive of a metastasis.

Anemia is marked. While the white count is high, the differential is not abnormal. The temperature is not mentioned, but I believe we may assume it was not remarkable. That the anemia is due to blood loss through the gastrointestinal tract is suggested by the strongly positive guaiac tests on the stools.

Without benefit of x-ray, one would have no hesitation in ascribing the right lower-quadrant pain, fatigue, loss of weight, and anemia to a carcinoma of the cecum. But in these days when that diagnosis can practically always be made by x-ray the failure to demonstrate any ulcerative lesion in this location must be considered significant. Is there any evidence that is opposed to this diagnosis? Right lower-quadrant pain usually signifies inflammatory rather than neoplastic lesions, but in a series of 100 consecutive cancers of the cecum that I have recently reviewed from this hospital, the commonest initial symptom was just this type of pain. The lack of change in bowel habits is not important, since a tumor in the cecum, not interfering with the fecal stream, will not cause any variation from normal. The high white count is not uncommon with ulcerative colonic lesions. Could the anemia be due to hypertrophic gastritis, suggested by the x-ray? If so, there should have been tarry stools in separate episodes rather than a steady loss of blood. Furthermore, the bleeding is not characteristic of hemorrhoids.

Evidence, despite the x-ray, therefore favors an

ulcerative lesion in the cecum. On this basis I shall exclude any form of appendicitis or inflammatory lesion such as regional ileitis. Tuberculosis of the cecum would be unusual with no evidence of pulmonary tuberculosis or calcified mesenteric nodes and with a negative tuberculin test. Malignant lymphoma is suggested by the palpable spleen and could account for the separate nodule by rectum. There are, however, no other enlarged nodes, and malignant lymphoma is primarily a submucosal lesion so that anemia is much less common with it than with cancer.

My diagnosis then, despite the lack of a positive x-ray diagnosis, is carcinoma of the cecum.

Preoperative Diagnosis

Carcinoma of the cecum?

Dr. Welch's Diagnosis

Carcinoma of the cecum.

Pathologic Diagnoses

Adenocarcinoma, Grade II, of the cecum with extension to the ileum.
Ileocecal fistula.
Meckel's diverticulum.

Pathological Discussion

Dr. Tracy B. Mallory: Preoperative diagnostic opinion on the ward ran along the same lines which Dr. Welch has covered in his discussion. Although a great variety of other lesions seemed possible, cancer of the cecum seemed much the most probable. Some thought was devoted to the possibility that the hypertrophic gastritis might have been the source of the gastrointestinal bleeding.

At operation still another possible source for hemorrhage into the intestinal tract was found, namely a Meckel's diverticulum. It, however, did not contain gastric epithelium and showed no evidence of ulceration. When Dr. Leland S. McKittrick explored the patient he found a large tumor mass in the right lower quadrant centering about the cecum and most extensive on its posterior wall. It had invaded the peritoneum and retroperitoneal tissues, particularly along the brim of the pelvis, and had involved the vas, which he was obliged to section. Both the terminal ileum and a portion of the transverse colon, which was very redundant, were adherent to the mass and had to be resected. He removed with the cecum all the ascending colon, half the transverse colon and about 40 cm. of terminal ileum, including the Meckel's diverticulum. He then did an ileotransverse colostomy. It was necessary to dissect fairly deeply into the retroperitoneal tissues over a wide area and some of the surfaces could not be peritonealized at the conclusion of the operation. The patient developed transient evidence of intestinal obstruction but despite this made a rapid and satisfactory convalescence.

When the specimen was examined in the laboratory it was found that the tumor had invaded directly through the cecal wall into the terminal ileum, which was extensively involved. There was a frank ulcer crater in the cecum, and in the center of this was a fistulous opening into the ileum. In spite of the extensive invasive growth, the regional lymph nodes appeared both grossly and microscopically to be free from tumor, and at operation no distant metastases were discovered.

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NEW ENGLAND POSTGRADUATE ASSEMBLY

At the June meeting of the Council of the Massachusetts Medical Society the Committee on Postgraduate Instruction recommended that a clinical congress or postgraduate assembly be sponsored by the Society sometime during the fall. This plan was approved by the Council, and the committee was instructed to work out the details.

As is well known, assemblies of this sort are regularly held in several of the large medical centers on the Atlantic seaboard. The outstanding example is the Graduate Fortnight, which has been held for many years under the auspices of the New York Academy of Medicine. Somewhat less pretentious but nonetheless important are the Postgraduate Institute of the Philadelphia County Medical Society and the Clinical Congress of the Connecticut State Medical Society. In each instance men of outstanding ability are the formal speakers, and as a rule, concurrent clinical exercises that cover the general topics under discussion are given. Such assemblies have proved to be of great value to physicians as a means of keeping "professionally fit," and attendance has by no means been limited to those from the immediate neighborhoods or even from the states in which they have been held. In each instance there has been a small registration fee to cover, in part, the expense.

In accordance with the instruction of the Council, the Committee on Postgraduate Instruction has made arrangements for a New England Postgraduate Assembly to be held at Sanders Theater, Harvard University, Cambridge, on November 15 and 16. Ten prominent medical educators and practitioners will deliver a series of thirty-minute talks covering a variety of topics of general interest in the diagnosis and treatment of disease. The assembly will start at ten on the morning of the first day and will end at five on the afternoon of the second; the program is given in detail on page vii of this issue of the *Journal*. The registration fee of three dollars covers admission not only to all sessions but also to an informal dinner in Memorial Hall, Harvard University, on the evening of the first day. Buffet luncheons for a nominal charge will be served in Memorial Hall each day.

All New England physicians are cordially invited to attend the assembly, and the program seems sufficiently broad in scope to attract a large number. Inasmuch as this is a new venture the committee is entirely in the dark in regard to the total registration — a figure of extreme importance in making the necessary arrangements. For the convenience of the readers of the *Journal* a registration coupon appears on page vii of this issue; all those planning to attend the assembly are earnestly requested to fill this out and to mail it at the earliest convenient moment.

NO RECESSION

THE *New York Times*, on September 20, prefaced an editorial discussion with the sentence: "Both physicians and the public cannot but approve the decision of the House of Delegates of the American Medical Association to recede from its previous position and, on the whole, to endorse the Government's program with the exception of the provision for compulsory health insurance." Had the *Times* ascertained what the previous position of the House of Delegates concerning the Government's program was, it could have found nothing from which to recede. The Government first announced its program in July, and the House of Delegates first expressed itself thereon at its special session on September 16 and 17. The House of Delegates gave a prompt and straightforward reply at the time of its first direct opportunity for self-expression. These facts are important in view of the present need for and apparent lack of medical leadership. A receding leadership is not a strong one.

Heretofore the Government has had little representative opinion from the medical profession. After the Atlantic City session in 1937, the resources of the American Medical Association were offered to the President. Probably because of lack of machinery these resources have not yet been utilized. Those who are primarily concerned with ancillary activities have been able to express their views, but those who are engaged in and indispensable to the proper medical care of the people have been unorganized as promoters and lobbyists.

It is to be hoped that the full text of the deliberations of the special session of the House of Delegates will be of such a nature that the profession as a whole may unite behind it and that the people as a whole may approve of the leadership thus provided.

MASSACHUSETTS MEDICAL SOCIETY

A stated meeting of the Council will be held in John Ware Hall, Boston Medical Library, 8 Fenway, Boston, on Wednesday, October 5, at 10:30 a. m.

BUSINESS:

1. Presentation of record of last meeting. (Published in *New England Journal of Medicine*, issue of June 30, 1938.)
2. Report of Committee of Arrangements.
3. Report of Committee on Membership.
4. Reports of other standing and special committees.
5. Reports of committees to consider petitions for restoration to the privileges of fellowship and appointment of new committees.
6. Appointment of an auditing committee.
7. Fill any vacancies in the offices of the Society.
8. Action on publication of the *Directory of Officers and Fellows*.
9. Incidental business.

ALEXANDER S. BEGG, *Secretary*.

Councilors are asked to sign one of the two attendance books before the meeting. The Cotting Luncheon will be served immediately after the meeting.

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

SPONTANEOUS RUPTURE OF THE UTERUS FOLLOWING PREVIOUS KERR CESAREAN SECTION

Mrs. S. M. M., a thirty-six-year-old gravida VI, at term, entered the hospital November 7, 1934, having been in labor sixteen hours.

The family history was non-essential. The patient's past history included the usual childhood diseases. In 1929 an appendectomy and a unilateral salpingo-oophorectomy were performed. There had been three previous miscarriages. In 1928 a Kerr cesarean section was performed and in 1932 a classical cesarean section was done because of the previous section. Catamenia began at thirteen and were regular every twenty-eight days. Her last menstrual period began February 3, making the expected date of confinement November 10.

Physical examination on entry revealed a healthy-appearing woman, who was complaining of labor pains. Her heart showed no enlargement; there were no murmurs. Her lungs were normal; there were no rales. The breasts showed the changes

A series of selected case histories by members of the section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the section.

of a multipara at term. The abdomen was filled with a tumor consistent with the size of a full-term pregnancy, and the fetal heart was heard. Uterine contractions were coming regularly at about three-minute intervals, and the cervix was two fingers dilated. The uterus was relaxing well between contractions.

Arrangements were immediately undertaken for a repeat cesarean section, but she became fully dilated before the operation could be started. Suddenly the patient complained of extreme abdominal pain, and the uterus became board-like. The maternal pulse quickly rose from 90 to 150, and the fetal heart rate dropped to 60. There was slight external bleeding. The diagnosis of rupture of the uterus was made, and laparotomy was immediately undertaken, an inlying catheter being inserted into the bladder before operation. At operation the visceral peritoneum over the surface of the uterus was intact, but a subperitoneal rupture was visible and extended transversely across the lower uterine segment in the region of the old Kerr scar. After incision of the peritoneum, a markedly asphyxiated infant was extracted through the rent. The placenta was expressed intact. The uterus contracted normally, and the blood loss was estimated at 300 cc. Inspection of the pelvic cavity showed that the indwelling catheter lay on the cervical stump. It was then evident that there was a large rent in the bladder extending from its dome posteriorly from left to right. The rent was approximately 15 cm. long and extended downward to within 3 cm. of the trigone. The edges of the rent were identified and closed with a continuous chromic-catgut suture, which infolded the bladder mucosa. A mushroom catheter was sutured into the dome of the bladder. Since there was a large hematoma in the right broad ligament, a supracervical hysterectomy was performed. After ligation and transection of the blood supply the uterus was amputated. The cervical stump was drained by means of a cigarette drain inserted downward into the vagina. The cervical stump was suspended by the round ligaments. Two drains were inserted down to the stump of the right broad ligament. The bladder was marsupialized extraperitoneally at the time of closure of the parietal peritoneum. The fascia was closed with No. 2 chromic catgut, and the skin with silk. At the close of the operation the pulse was 120, and the blood pressure 84 systolic, 40 diastolic. The baby died shortly after birth.

The patient's convalescence was smooth and entirely afebrile. The vaginal drain was removed on the ninth postoperative day, and the abdominal drain on the twelfth day. The suprapubic catheter was removed on the fourteenth day, and the urethral catheter on the twenty-first day. She had a bladder residual up to 400 cc. for a few days but was discharged on the twenty-sixth postoperative day without a residual, and perfectly continent.

Comment. The low section was originally advocated because it presumably did away with the possibility of subsequent ruptures. This case proves that such an assumption is not true. Due to distention as pregnancy advances, the classical section ruptures more frequently before labor; the low section usually ruptures under the stress of labor. Laparotomy is the operation of choice, since no other operation meets the conditions. The rupture of the bladder reported in this case is fortunately not a common accompaniment of uterine rupture. There is no mention in this case of transfusion; it probably was done. It certainly is a very important part of the treatment of these cases and should be resorted to very freely after cross-matching has taken place.

DEATHS

ELY — Theodore W. Ely, M.D., of Central Street, Manchester, died September 25. He was in his fifty-fourth year.

A native of Elyria, Ohio, he received his degree from Western Reserve University School of Medicine in 1909 and later served as an intern at the Children's Hospital and the Boston City Hospital. He was a member of the medical corps of the 26th Division and served in France with that unit. After the War he was appointed medical inspector at the Boston immigration station where he served for a year. He conducted a private practice in Bedford before going to Manchester.

A member of the American Medical Association and the Massachusetts Medical Society, he was also a member of the University Club of Boston.

His widow and a sister survive him.

HOOD — Mary G. Hood, M.D., of 20 Charlesgate West, Boston, died September 19. She was in her eighty-ninth year.

Born in Bath, New York, she received her degree from the Woman's Medical College of Pennsylvania in 1874. The year following she served as assistant resident physician of the Woman's Hospital in Philadelphia. In 1876 she was made chief medical officer of the Nursery and Child's Hospital of New York and the following year she became teacher of physiology and resident physician at Wilson College in Chambersburg, Pennsylvania. Dr. Hood started private practice in Minneapolis, Minnesota, in 1878. During seventeen years there she founded Bethany Home and the Northwestern Hospital for Women and Children. She came to New England in 1895 and practiced in Newton Center until her retirement in 1914.

She was a member of the American Medical Association, the Massachusetts Medical Society and the Medical Women's National Association.

A sister survives her.

VERMONT STATE MEDICAL SOCIETY

DEATH

McSWEENEY — PATRICK E. McSWEENEY, of Burlington, graduated from the University of Vermont School of Medicine in 1886 and was professor of obstetrics and gynecology there from 1914 to 1937. He was an attending surgeon at the Mary Fletcher Hospital, Bishop DeGoesbriand Hospital and Fanny Allen Hospital in Burlington and the Porter Hospital in Middlebury, and consulting gynecologist at the Mary Fletcher Hospital.

His offices included the presidency of the Chittenden County Medical Society in 1902 and of the Vermont State Medical Society in 1905 and the chairmanship of the Vermont State Society for the Control of Cancer.

He was the author of *The Cancer Problem in Vermont* and editor of the cancer publicity bulletin. Dr. McSweeney received an honorary degree of Master of Science from the University of Vermont in 1902. He was a fellow of the American College of Surgeons, a member of the Vermont State Medical Society, the New York and New England Surgical societies, the American Medical Association and the American Society for the Control of Cancer, and a trustee of the Vermont School for Feeble-Minded.

He died September 2, at Burlington, at the age of seventy-eight.

MISCELLANY

SOME FUNDAMENTALS IN TUBERCULOSIS PREVENTION

"To the student of tuberculosis there would appear to be conflicting opinions in the minds of those directing anti-tuberculosis campaigns in different countries and states in recent years. This confusion is more apparent than real, and arises from the fact that different countries are featuring different majors while pursuing a general program. . . . For health and tuberculosis workers at this time nothing could be more constructive than some thread of knowledge that would lead to a better understanding of the values and limitations of these methods of control."

With the above in mind Dr. R. G. Ferguson (Some Fundamentals in Tuberculosis Prevention. Canad. Pub. Health J., 29:203-212, 1938.) proceeds to analyze the strengths and weaknesses of the various methods for the control of tuberculosis in a paper which will be read with interest by all students of tuberculosis, and which contains some excellent suggestions for the general practitioner.

NON-SPECIFIC RESISTANCE

The importance of general resistance was well established before tuberculosis was known to be a germ disease; widespread tuberculosis was found associated with poverty; better living conditions provided some measure of protection.

Therefore in a community where the disease is endemic, and where the tuberculosis death rate is high, a good standard of living is excellent general treatment.

However, this has its limitations: it does not prevent infection. It gives inadequate protection to the non-resistant and cannot protect even the resistant against large and frequent doses of infection.

In areas where the death rate is low and infection no longer inevitable it is giving way to more direct measures aimed at the infectious nature of the disease.

SANITATION

The anti-tuberculosis program has since its inception stressed sanitary education and undoubtedly infection has been reduced as a result.

Nevertheless the protection conferred by sanitary habits is in a practical way also limited. It is acquired after long and intensive practice, is maintained at the price of eternal vigilance and is subject to human error.

ELIMINATION OF INFECTION

Perhaps the greatest benefit conferred by the modern sanatorium movement is not the lowering of the death rate by cures but the lowering of the infection rate by segregation and isolation which has perhaps given us the key to the ultimate control and eradication of this disease.

Unlike the acute respiratory diseases which depend for their spread on many cases being infectious for a short period, tuberculosis is a more slowly developing infection and gives much more time to isolate it.

Isolation is now the most effective measure for the control of tuberculosis, but in order for it to be effective there must be (1) ample bed accommodation, (2) the removal of financial barriers to treatment without flavor of charity, (3) the most efficient treatment procurable provided for all, and (4) institutions sufficiently comfortable to be acceptable to patients for indefinite periods.

VACCINATION

Any assistance that could be secured from even a relatively successful prophylactic would be of great help, and we should be open minded with regard to BCG.

EPIDEMIOLOGICAL STUDIES

As the death rate declines programs must become more selective, concentrating on those groups where infection is heaviest and which can only be determined by continuous epidemiological studies in the area under control. Such studies indicate the strength of the enemy and the location of concentration or weaknesses, and provide information for a plan of attack.

MORE CAREFUL SIFTING OF CONTACTS AND SUSPECTS

The clearing up of infection depends in many cases on the interest and energy of the family physician.

Where the incidence of infection is low and where the people are tuberculosis-conscious, the next step appears to be the supplying of free tuberculin to the family physician and his familiarization in its use as an aid in case selection.

UNIDENTIFIED SPREADERS

The greatest difficulty in clearing up tuberculosis is the infectious person with good tolerance who may spread the disease for years before falling sick. These persons appear to account for more than half the new patients admitted to sanatoriums even where an advanced program is applied.

How to identify the near-well, chronic spreader, infectious, but not sick enough to report to a doctor, is the difficult problem in tuberculosis epidemiology.

One simple suggestion toward its solution is a more general use of sputum examination by the family physician.

It is not too much to expect that the family physician should take the responsibility of having the sputum of chronic coughers in his practice examined for tubercle bacilli.

There is perhaps no case-finding procedure a physician can follow which will yield higher returns for the same effort.

Sputum examinations of chronic coughers would succeed in measurably reducing infection from now unidentified spreaders.

FOLLOW-UP OF EX-PATIENTS

No program is complete which does not give due consideration to the re-examination of all ex-patients for an average period of four years after discharge. This re-examination is not only for the purpose of advising and assisting them to attain the greatest possible degree of recovery but is also for the purpose of selecting cases which become active and infectious and which require further treatment for recovery and segregation for prevention of infection.

CASE REGISTRATION

The greatest flaw in the armor of anti-tuberculosis work today is failure to accomplish adequate case registration in the absence of which systematic follow-up of either ex-patients or contacts is impossible.

Registration cannot be achieved by legislation alone and as a statistical effort alone will fail. It must, to be successful, include an active follow-up service which provides advice, examination and treatment, if necessary, for patients and their contacts.

TUBERCULOSIS-CONSCIOUSNESS

A fundamental of the anti-tuberculosis program upon which in the end all other tuberculosis activities depend is the tuberculosis-consciousness of the people.

The ailing individual must initiate the first step and come to the doctor.

When will he come? If he comes only on falling sick the great majority will appear in an advanced stage of the disease; if on suspicion of early disease a large proportion will come in the early stage. How can he come on suspicion unless he has been taught to suspect tuberculosis?

While health officers, health nurses, family physicians, clergy, ex-patients, radio and press can accomplish a great deal, the responsibility could be shared with many thousands of fully trained teachers and "there appears no reason why health cannot be taught in school as successfully as can the three R's."

In a tuberculosis-minded community with modern facilities for diagnosis, tuberculosis can be diagnosed early enough and isolated early enough to reduce the spread of infection so rapidly as to convince us that it can be controlled and eventually reduced to a very minor cause of death. — *Tuberculosis Abstracts* (September, 1938).

NOTICES

FAULKNER HOSPITAL CLINICOPATHOLOGICAL CONFERENCES

The clinicopathological conferences at the Faulkner Hospital during the coming year will be held, as usual, at five o'clock on the first Tuesday afternoon of each month. At each, two or three interesting cases will be discussed and diagnosed by physicians who are unfamiliar with them, and the pathological findings will then be presented.

The first meeting of this season will be held on October 6. Dr. William B. Breed and Dr. Herbert L. Johnson will discuss the cases.

INTERNATIONAL CONGRESS OF MILITARY MEDICINE AND PHARMACY

The forthcoming International Congress (10th) of Military Medicine and Pharmacy will be held in Washington, May 7 to May 15, 1939. Invitations to participate have been sent to every country by the President of the United States. General Charles R. Reynolds, surgeon general of the United States Army, will be the president of the congress. Registration is open to all officers of the medical services of the army, navy, air and colonial services, national guard, territorial forces and public health service, whether active or reserve.

Colonel Harold W. Jones of the United States Army Medical Corps is secretary general. The secretarial office is at the Army Medical Library, Washington, D. C.

BOSTON DISPENSARY

25 Bennet Street, Boston
Lecture Hall, Second Floor, 9-10 a. m.

MEDICAL CONFERENCE PROGRAM, OCTOBER, 1938

Tuesday, October 4 — Differential Diagnosis of Certain Lesions of the Spine. Dr. J. D. Adams.
Wednesday, October 5 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, October 6 — Certain Aspects of the Endocrine Treatment of Menopausal Symptoms. Dr. C. H. Lawrence.
Friday, October 7 — Acute and Chronic Mediastinitis. Dr. C. S. Keefer.
Saturday, October 8 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, October 11 — Presentation of Dermatological Cases. Dr. F. M. Thurmon.
Thursday, October 13 — Blood Clinic. Dr. I. Olef and Dr. H. G. Brugsch.
Friday, October 14 — The Nature, Criteria of Treatment and Prognosis of Multiple Sclerosis. Dr. T. J. Putnam.
Saturday, October 15 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, October 18 — Clinicopathological Conference. Dr. R. C. Wadsworth and Dr. William Dameshek.
Wednesday, October 19 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, October 20 — The Mechanism of Rupture of the Graafian Follicle. Dr. J. T. Smith.
Friday, October 21 — The Immediate and Ultimate Prognosis of Heart Disease. Dr. P. D. White.
Saturday, October 22 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Tuesday, October 25 — X-ray Demonstration. Dr. Alice Ettinger.
Wednesday, October 26 — Hospital Case Presentation. Dr. S. J. Thannhauser.
Thursday, October 27 — Cancer of the Lung. Dr. Richard Overholt.
Friday, October 28 — Metabolism of Bone. Dr. J. C. Aub.
Saturday, October 29 — Hospital Case Presentation. Dr. S. J. Thannhauser.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, OCTOBER 3

TUESDAY, OCTOBER 4

Massachusetts Medico-Legal Society, Mallory Institute of Pathology, Boston City Hospital.

*9-10 a. m. Boston Dispensary. Differential Diagnosis of Certain Lesions of the Spine. Dr. J. D. Adams.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

WEDNESDAY, OCTOBER 5

Massachusetts Medico-Legal Society, Mallory Institute of Pathology, Boston City Hospital.

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*11:30 a. m. - 12:30 p. m. Psychiatric Conference. Boston City Hospital. Nerve Conference Room, 6th floor, Out-Patient Department.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

THURSDAY, OCTOBER 6

*9-10 a. m. Boston Dispensary. Certain Aspects of the Endocrine Treatment of Menopausal Symptoms. Dr. C. H. Lawrence.

*10:30 a. m. Psychiatric Rounds. Boston City Hospital. Rounds will start at Room 916 on the ninth floor of the Medical Building.

*5 p. m. Faulkner Hospital, clinicopathological conference.

FRIDAY, OCTOBER 7

*9-10 a. m. Boston Dispensary. Acute and Chronic Mediastinitis. Dr. C. S. Keefer.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

12 m. Clinical meeting of the Children's Medical Service, Massachusetts General Hospital, Ether Dome.

SATURDAY, OCTOBER 8

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

OCTOBER 3-15 — Inactive Status Training Course. Page 288, issue of August 25.

OCTOBER 4 — Lawrence Cancer Clinic. Page 450, issue of September 22.

OCTOBER 4 and 5 — Massachusetts Medico-Legal Society. Page 365, issue of September 8.

OCTOBER 4-29 — Boston Dispensary. Medical Conference Program. Page 501.

OCTOBER 6 — Faulkner Hospital, clinicopathological conference. Page 501.

OCTOBER 7 and 8 — Conference on Rural Medicine. Page 180, issue of August 4, and page 407, issue of September 15.

OCTOBER 8 and NOVEMBER 15 — American Board of Ophthalmology. Page 282, issue of February 10.

OCTOBER 13 — Pentucket Association of Physicians, 8:30 p. m., Hotel Bartlett, 95 Main Street, Haverhill.

OCTOBER 17, 1938 and FEBRUARY 20, 1939 — American Board of Internal Medicine, Inc. Page 216, issue of August 11.

OCTOBER 17-21 — Clinical Congress of the American College of Surgeons, New York City.

OCTOBER 24-26 — Academy of Physical Medicine, Scientific Session. Washington, D. C.

OCTOBER 24 - NOVEMBER 4 — Graduate Fortnight of the New York Academy of Medicine. Page 181, issue of August 4.

OCTOBER 25-28 — American Public Health Association. Page 321, issue of September 1.

OCTOBER 31, NOVEMBER 1, 2, 3 and 4 — Inter-State Postgraduate Medical Association. Page 451, issue of September 22.

NOVEMBER 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

NOVEMBER 5, 1938; FEBRUARY 4, MAY 15 and 16, 1939 — American Board of Obstetrics and Gynecology. Page 451, issue of September 22.

NOVEMBER 15-16 — New England Postgraduate Assembly. Page vii, Advertising Section.

MARCH 27-31, 1939 — American College of Physicians. Page 36, issue of July 7.

MAY 7-15, 1939 — International Congress of Military Medicine and Pharmacy. Page 501.

SEPTEMBER, 1939 — Boston Psychoanalytic Institute. Page 450, issue of September 22.

BOOK REVIEWS

The Horse and Buggy Doctor. Arthur E. Hertzler. 322 pp. New York and London: Harper & Brothers, 1938. $2.75.

This is a fine autobiography of a self-made Kansas doctor. His education both in this country and Europe, his struggles as a general practitioner and later as a surgeon, the establishment of his own splendid hospital in Halstead, Kansas, and his superb attitude toward medicine, research and the public are all outlined, clearly, pungently and yet with a sense of balance and justice seldom found in our fellow men. If one wants to visualize the real background of medicine in this country and learn what manner of men we are, or ought to be, one should read Dr. Hertzler's book. It is a sound tonic to be taken in generous doses. Turn from the bickerings of the few, the committee reports and the "socialization menace" and find out what one good American could do to educate himself and serve the public with a type of medicine that makes us all proud of the profession in this country. His last chapter, "Medicine as It is Today," is not to be missed by any physician, for a sounder piece of writing on the general subject of medicine has seldom come to the reviewer's attention.

Medical State Board Questions and Answers. R. Max Goepp. Seventh edition. 644 pp. Philadelphia and London: W. B. Saunders Company, 1938. $5.50.

This book presents an elaborate collection of questions submitted to applicants for registration as physicians with appropriate answers. The technical departments of medicine are well covered, and there are well-selected references to many problems before practitioners so that it may serve as a vade mecum in overcoming lapses of memory.

Fortunately, there is no evidence of the author's expectation that well-informed persons will be likely to infer that this volume is quite up to date in dealing with general medicine, because it is widely recognized that no book can keep pace with the advances constantly under way.

It is well written and arranged and will undoubtedly help those undergoing the written tests required by examining boards.

A Text-Book of Pathology: An introduction to medicine. William Boyd. 1064 pp. Philadelphia: Lea & Febiger, 1938. $10.00.

This edition of Boyd's textbook will maintain its outstanding popularity with students in spite of its increased size, which now verges on the ponderous.

Frankly for the student rather than the practitioner, it is written with a pleasant, clear, rather dogmatic style. The 459 figures are well selected and technically excellent. There are 16 colored plates, 8 of them new, which will be of great help to the student.

It keeps pace amazingly well with recent work and includes a discussion of the Shwartzman phenomenon, the Shope papilloma, experimental carcinogenesis, the effect of radiation on tumors, and other useful points not often found in a pathological text.

If occasionally there is a trace of excessive emphasis, as in the failure to make clear all the limitations of tumor grading, this is more than compensated for by the clear, well-smoothed trail hewn through the maze of pathological detail.

The Radiology of Pulmonary Tuberculosis. J. E. Bannen. 156 pp. Baltimore: William Wood & Company, 1937. $4.50.

This is an excellent brief review of clinical and x-ray findings in pulmonary tuberculosis. There are only about 69 pages of text, 87 pages being given over to illustrations which are produced strikingly well. It is succinctly written. If anything, there has been too great compression of material, perhaps to keep the price of the volume within reason. Much more could have been said, and properly, on the differential diagnosis of silicosis, asbestosis, infarct and new growth, which have assumed such importance in recent years. It is to be hoped that the author will expand the present volume in the near future.

Illustrations of Anatomy for Nurses. E. B. Jamieson. 62 pp. Baltimore: William Wood & Company, 1938. $3.00.

Those who are already familiar with Jamieson's *Illustrations of Regional Anatomy* for students and practitioners of medicine and surgery will be pleased to learn that sixty-two of the plates have been suitably arranged for nurses in such institutions where the teacher recommends a supplement to the regular textbook. The high character of these illustrations deserves strong recommendation for collateral study.